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SOME THEORETICAL CONSIDERATIONS CONCERNING CHLOROFORM.*

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Chloroform, once the most widely used anesthetic substance, has now been generally discarded in this country, although it is still widely used in Europe. In view of the newer and safer anesthetic methods, such as ether, nitrous oxid, and local anesthesia, there is now little or no justification of its use. Nevertheless, for experimental purposes it is of great interest in providing a means of investigating not only the fundamental question of what the phenomenon of anesthesia is but also the question of the origin of certain changes in the tissues which accompany to a more or less degree the use of every anesthetic agent. It is a consideration of these changes which I wish to dwell upon in this article.

It has long been known that after a prolonged administration of chloroform a condition, often fatal, may manifest itself from one to three days which has been designated as *"Late Chloroform Poisoning."* This condition is characterized clinically chiefly by uncontrollable vomiting, the presence of excessive quantities of acetone, aceto-acetic acid and increase of nitrogen in the urine, marked mental symptoms of drowsiness and coma either with or without preceding delirium, multiple ecchymotic hemorrhages and occasionally icterus. At autopsy edema, fat infiltration of the viscera, multiple hemorrhages and necrosis of the central portion of the liver lobule, are conspicuous features.[1]

There has never been an adequate explanation of these phenomena. Yet such an explanation would be of the utmost importance, since the same changes are found accompanying intoxications with a large number of agents, including other narcotic substances, arsenic, salvarsan, phosphorus and most, if not all, bacterial poisons. It is unnecessary to emphasize the importance of even a partial explanation of not only how the narcotics but also how bacteria produce their poisonous affects.

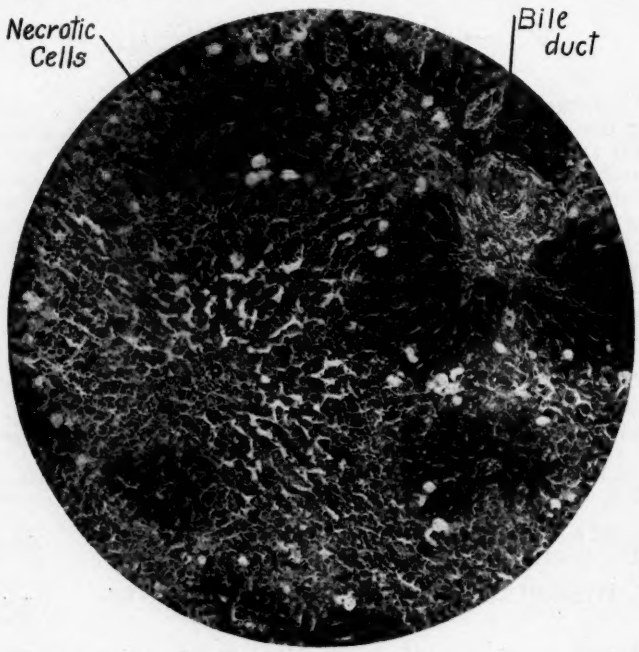

Fig. 1. Typical central liver necrosis produced by chloroform anesthesia for four and one-half hours in Dog B of Experiment 3. Nearly all the lobule is affected. Microphotograph. X 83.

In a recent article[2] the author has shown that at least in the case of chloroform poisoning the evidence is strong that not only the necrosis of the liver but also the fat infiltration, the tendency to the production of hemorrhages and the edema are acid effects. With chloroform the responsibility probably rests chiefly upon hydrochloric acid which is formed during the destruction of chloroform within the body. Similarly, alkyl halides in general not only produce these changes but are also capable of yielding halogen acids (hydrochloric, hydrobromic, hydriodic, as the case may be) in their met-

*Read during the Joint-Session of the Interstate Association of Anesthetists and the National Dental Association, at Louisville, Ky., July 26-27, 1916.

abolic break-down. These changes, however, are not limited to intoxications with those substances which can split off mineral acids, but they may follow the administration of any substance that may cause tissue asphyxia with its attendant accumulation of organic acids. Some of the evidence favoring this idea will be presented here, but for most of the details the original article must be consulted.

The fact that these changes are readily obtained with chloroform and only with difficulty by ether suggests that the difference in behavior of the two anesthetic substances must be due either to a difference in molecular action or to a difference in the products formed during their breakdown. Now it is interesting that when chemically pure chloroform is exposed to the sun-light in the presence of oxygen, hydrochloric acid and phosgene are formed, as expressed thus:

$$CHI_3 + \leftrightarrows HCI + COC_2$$

This oxidation occurs so easily that it has become necessary for manufacturers of chloroform for anesthic purposes to add alcohol to it to prevent decomposition. Phosgene in the presence of water is really decomposed into two molecules of hydrochloric acid and one of carbon dioxid thus:

$$COC_2 + H_2O \leftrightarrows 2HCI + CO_2$$

Therefore it is apparent that for every molecule of chloroform which is oxidized in the presence of water three molecules of hydrochloric acid are formed. Furthermore this decomposition of chloroform is so easily performed that it occurs spontaneously. It is thus reasonable to suppose *a priori* that there is no reason why the decomposition might not occur under the conditions met with in the body, where there is available oxygen and available water. It is not difficult to imagine that the liberation within the body of so strong an acid as hydrochloric might induce severe disturbances.

With these facts and considerations in view, the hypothesis was subjected to a series of experimental tests which may be outlined as follows:

(1) A study was undertaken of the morphological changes induced by hydrochloric acid, with special reference to the liver, since that is the organ which always shows to the most extreme degree the changes mentioned above. (2) Attempts were made to demonstrate free hydrochloric acid in the necrotic areas in the livers of animals poisoned with chloroform. (3) Observations were made on the relative powers to produce these changes of different chlorine substitution products of methane (e.g., dichlormethane (CH_2 CI_2), chloroform ($CHCI_3$), and tetrachlormethane (CCI_4), which on theoretical grounds could be considered to yield different amounts of hydrochloric acid in their breakdown. (4.) The inhibiting effect of alkali was studied. (5.) Attempts were made to produce the typical picture of chloroform poisoning by other alkyl hal-

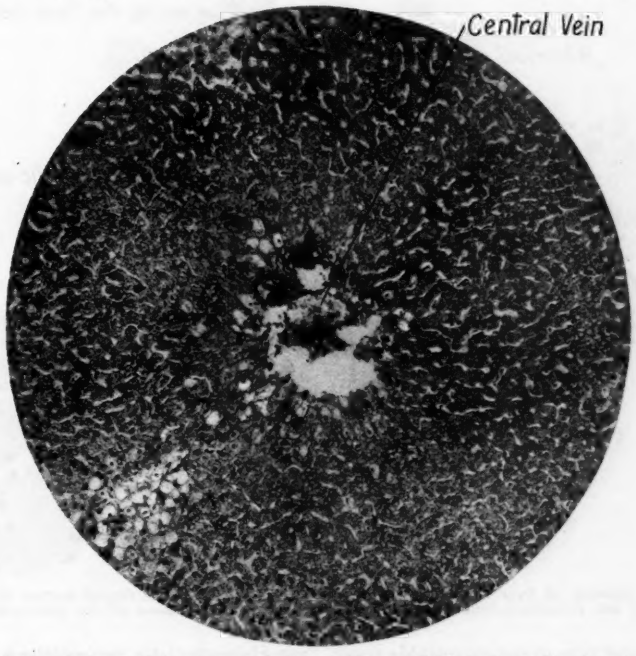

Fig. 2. Inhibition of necrosis by the intravenous injection of 150 cc. of Fischer's alkaline hypertonic salt solution as shown in Dog A of Experiment 3, which had received chloroform for the same length of time as Dog B (Fig. 1). There is practically no necrosis. Only a slight amount of fat accumulation in the cells about the central vein has occurred. Microphotograph. X83.

ides of the same type as chloroform, viz., bromoform ($CHBR_3$), and iodoform (CHI_3), which might be expected to give analogous products in their breakdown, with, however, the liberation of hydrobromic acid and hydriodic acid, respectively, instead of hydrochloric acid. (6.) Experiments were made to ascertain whether or not morphological effects like those produced by chloroform can also be induced by alkyl halides in general, and whether these substances are decomposed in the body in such a way that the corresponding halogen acid is liberated. (7) A comparative study of the lesions produced by chloral hydrate (CCl_3—CHOH) and chloroform ($CHCl_3$) acid in its metabolic breakdown, although it contains the same number of chlorin atoms as chloroform.

monizes with the frequency of their occurrence after tissue asphyxia, with its attendant formation of acid. In a previous paper in which it was shown that the various hemorrhage diseases of the newly born are probably expressions of an asphyxial process, I stated that this hemorrhagic tendency might be due to a *"more fundamental and wide-spread change, as a result of which not only fibrinogen, but innumerable other proteins tend to remain in solution, with the result that apart from diminished blood vessels walls." The production of fatty changes in the liver with hydrochloric acid is in accordance with an observation of Leathes.* It is of great fundamental interest that, as shown here, by the use of a single experimental agent (hydrochloric acid) all those changes in the liver may be produced which

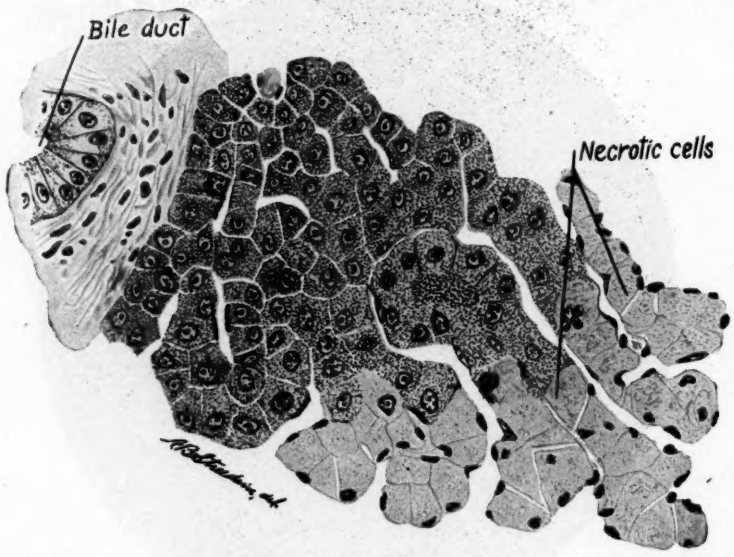

Fig. 3. High power drawing of liver shown in Fig. 1. The necrotic cells in the central portion of the lobule stain intensely with eosin. The nuclei of the parenchymatous cells have disappeared; and only those of the capillaries are evident. X356.

When hydrochloric acid was given in suitable concentration, to rabbits and dogs either by mouth or by injection into a branch of the portal vein it induced all the changes in the liver which are characteristic of chloroform poisoning except that the necrosis was at the peripheries instead of at the centers of the lobules. This difference in location of the necrosis was to be expected by the nature of the experimental conditions, since, owing to the arrangements of the blood supply, the acid entered the lobule at the periphery and therefore exerted its maximum effect at that location. The extensive edema which follows the administration of hydrochloric acid tends to confirm the well known observations of Martin H. Fischer[8] on the imbition of water by tissue colloids under the influence of acids generally. The production of hemorrhages are characteristic not only of chloroform poisoning but also of bacterial action and intoxications with many other substances.

Attempts to recognize the presence of free hydrochloric acid in the livers of animals which had been subjected to a deep chloroform poisoning were carried out in the following way: Guinea-pigs which two days previously had been subjected to a deep chloroform anesthesia for about four hours consecutively were anesthetized with ether and while still alive portions of the liver were removed, cut into thin sections very quickly by means of a Valentine knife and immersed in baths of different indicators on the one hand, and in baths of silver nitrate on the other. The only way by which free hydrochloric acid can be detected is by recognizing the presence of free hydrogen ion (H+) and free chlorine ion

(Cl). For the purpose of detecting free hydrogen the best indicator was found to be neutral red; and by means of this, in some of the pigs a hydrogen ion content as high as 10^6 was noted, which greatly exceeds the amount ever reached in the tissues under normal conditions. For detecting free chlorin ion, the method of Macallum and Menten was used of observing the blackening of the sections after treatment with silver nitrate and nitric acid. By means of these two methods it was found that the necrotic areas of the liver lobules contained free hydrogen ion and free chlorin ion. The presumption therefore was strong that these areas contained free hydrochloric acid. These results, however, cannot be considered conclusive evidence of the presence of free hydrochloric acid, since certain other conditions may be present to complicate the results. These are more amply discussed in the original article.

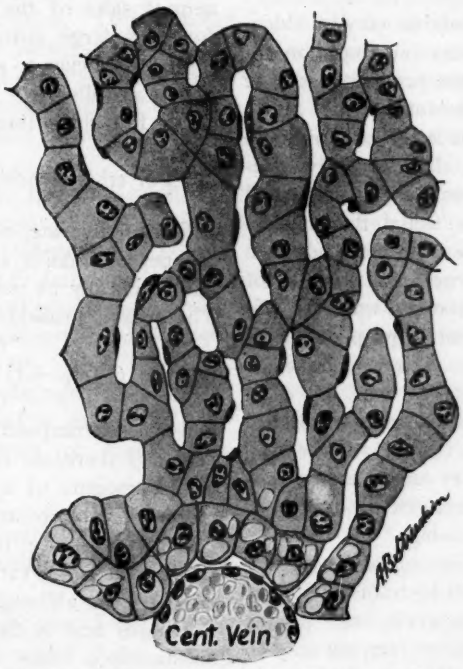

Fig. 4. High power drawing of the liver shown in Fig. 2. There is no appreciable necrosis; and only a moderate accumulation of fat has occurred in the parenchymatous cells around the central vein. X356.

A study of other chlorin substitution products of methane should be of interest from the standpoints of determining (1) whether they all have the power of producing changes in the tissues identical with those of chloroform, and (2) whether this property is proportional to the number of molecules of hydrochloric acid which could theoretically be derived from them. Chloroform, of course, is the tri-chlorin substitution product of methane. If, starting with methane (CH_4), we should outline a series of its various chlorine substitution products, we might expect, according to the theory, that those which could give the largest amounts of hydrochloric acid in their breakdown would manifest the strongest tendencies to produce the necrosis and other changes. Thus *a priori* we might expect the series to run in this order:

$$CH_3Cl+CH_2Cl_2+CHCl_3+CCl_4,$$

If all these substances were equally broken down if all these substances were equally broken down in the body. The last three of the series (viz., CH_2Cl_2, $CHCl_3CCl_4$), were selected experimentally as sufficient to test the tenability of this hypothesis. Outside of the body it had already been known that CCl_4, when heated with water can yield four molecules of hydrochloric acid and CH_2Cl_2 can yield two. That chloroform can yield three molecules of hydrochloric acid in its breakdown has already been shown above. When these various substances were administered to animals by innhalation, it was found that not only did all three possess the power to produce edema, hemorrhages, fatty changes in the organs and central necrosis of the liver, but that this power was shown in greatest degree by tetrachlormethane (CCl_4) and least by dichlormethane (CH_2Cl_2) A comparison of the minimum fatal doses of the three substances when given intravenously to rabbits again bore out the

same relationship. The following table shows the minimum fatal doses:

Substance	Minimum fatal doses per kilo. of rabbit.	Minimum fatal doses expressed in gram-molecular contrations.
CCl_4	0.053 gm.	0.000344
$CHCl_3$	0.085 gm.	0.000711
CH_2Cl_2	0.147 gm.	0.00161

Thus it is evident that the power of these various substances to produce the extensive tissue changes paralleled the amounts of hydrochloric acid which they can give in their respective breakdowns outside of the body.

Alkali, if simultaneously administered with chloroform and in suitable concentration, ought to inhibit at least to some extent the production of the various lesions induced by chloroform if they are to be considered as chiefly acid effects. This was found to be the case. When alkali was given intravenously to dogs simultaneously with a protracted chloroform anesthesia, it was found that it protected the dog to a large degree from the production of the changes. Fischer's hypertonic solution of sodium chlorid and sodium carbonate was very efficient in this respect when given in proper concentration. The illustrations show this protective action of the alkali.

Iodoform (CHI_3) and bromoform ($CHBr_3$), are so similar to chloroform ($CHCl_3$) in chemical structure as to suggest that they might have a similar power to produce the characteristic morphological changes of chloroform poisoning.

Here, however, we should of course be dealing with an effect of hydriodic and hydrobromic acids, respectively, instead of hydrochloric acid, by the terms of the theory. As a matter of fact, not only do both of these substances induce lesions which are in every way identical with chloroform effects, but it is also possible to obtain some evidence that in each case the respective halogen acid (HBr and HI) is produced in the body. Striking evidence of this is found in the fact that neutral salts of these acids are found in large qaantities in the urine of animals poisoned with bromoform and iodoform. Also by examination of the necrotic areas of the livers of such animals with methods similar to those previously described in connection with chloroform, it was found that these areas contained a large amount of free hydrogen ion and were therefore strongly acid in reaction.

The readiness with which all of the halogen substitution products of methane induced the changes made it seem likely that the same results would follow the use of similar substances of the ethane group. Such was found to be the case with each substance used. The other substances used were ethyl chlorid, ethyl bromid, ethyl iodid and ethylene bromid. Accordingly the results suggest that the power to produce edema, hemorrhages, fatty changes in the viscera and central necrosis of the liver lobules is a property of all those chemical substances which belong in the group of aliphatic alkyl halides. In connection with the theory being developed here, it is very interesting and suggestive that all aliphatic alkyl halides are capable of yielding halogen acids in the test-tube. That they do so also in the body seems to be shown by the fact that after poisoning with all of the substances which were tried, the neutral salts of the respective halogen acids are found in large quantities in the urine. Nef has brought evidence to show that the type of dissociation is as follows:

For chloroform, $CHCl_3 \rightleftarrows CCl_2 + HCl$

For ethyl chlorid, $C_2H_5Cl \rightleftarrows CH_2CH + HCl$

Chloral hydrate, although it possesses the same number of chlorin atoms as does chloroform, yet has practically no power to produce these changes. The reason probably lies in the fact that chloral hydrate ($CCl_3—CH\begin{matrix}OH\\OH\end{matrix}$) in its breakdown in the body is decomposed into trichlorethylglucuronic acid and therefore is incapable of forming appreciable amounts of hydrochloric acid in contradistinction to chloroform.

Other substances to a less extent have the power of producing the various tissue changes under discussion even although they have no power to yield a halogen acid in their breakdown. One of these substances is ether. But ether, like all anesthetic agents, interferes with the normal exchange of the cell and therefore induces a tissue asphyxia. In any condition of tissue asphyxia, no matter by what means produced, there is an increased production and accumulation of organic acids. Probably, therefore, the syndrome of edema, fatty changes, the tendency to the production of hemorrhages, and necrosis is always an expression of the effect of an acid or of some agent which acts like an acid on the tissues.

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CONTROL OF CIRCULATORY DISTURBANCES UNDER ANESTHESIA AND ANALGESIA.*

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INTRODUCTION.—It would hardly seem necessary to mention the importance of carefully watching the pulse changes during anesthesia. But to be told in certain clinics that the practice is to *"disregard the circulation and watch only the respiration"* would indicate that it is necessary to emphasize the importance of watching the circulation. Pulse changes are harder to interpret than respiratory changes and this fact may explain why some so-called *anesthetists* ignore the pulse. To ignore the circulation is to overlook one safeguard in the successful administration of anesthetics. Blood-pressure estimations aid one materially in warning one of approaching shock, and such estimations should be undertaken in most cases.

CAUSES FOR CIRCULATORY DISTURBANCES.

There are numerous predisposing factors in the causation of circulatory phenomena, such as profound anemia, hypertrophied or fatty heart, mental exhaustion, and struggling during induction. Getting at the real causes we may classify them under four headings:

(a) over-dosage of anesthetic agent;
(b) asphyxia (either partial or complete);
(c) shock (either vaso-motor or cardio-inhibitory), and
(d) hemorrhage.

It is vitally necessary to determine definitely the cause for the circulatory disturbances before proper treatment can be applied.

OVER-DOSAGE OF ANESTHETIC AGENT.

Chloroform most assuredly and without warning may cause grave circulatory disturbance. For this reason I have for a number of years practiced continuous auscultation in all of my chloroform cases. I have records of several cases indicating the great value of the phonendoscope in showing the approach of circulatory trouble.

In healthy subjects nitrous oxid will not produce primary circulatory disturbance, but in subjects with valvular or other cardias disease this may occur. Nitrous oxid, if given pure and by a closed method, will rapidly produce anoxemia and secondarily circulatory disturbance.

The plan of administering gas-ether should be relegated to the past and a gas-oxygen-ether sequence substituted. In fact, I believe that nitrous oxid should never be used as an anesthetic unless a sufficient supply of oxygen is on hand.

Ether resembles nitrous oxid in that primary circulatory disturbances never occur when it is used in healthy subjects. In subjects with marked valvular defects, with fatty hearts or with grave myocardial conditions ether may cause circulatory symptoms, but even here the symptoms ordinarily follow slight asphyxia. When respiratory failure occurs under ether the circulation will maintain itself sufficiently long for proper measures to be applied.

ASPHYXIA.

A very frequent cause for circulatory disturbance is asphyxia, and it should be the constant aim of the anesthetist to maintain an open air-way. Artificial air-ways are of great value and should be on hand at all times. Vomiting during induction may tend to produce temporary asphyxia, and when this occurs the plan to follow is to keep the head turned to one side, to retain the mask in position and continue the anesthetic.

Faulty position may contribute to partial asphyxia and this is especially true in operations for laminectomy. Here the anethetist must be constantly on the alert for asphyxial symptoms. The plan I find effective is to elevate one shoulder with pads or with a brace, to lower the head, turning it to the side elevated by the pads.

Very obese patients have an inherent tendency to asphyxia due to the lessened breathing capacity in proportion to body weight. With them my rule is to employ an oxygen-ether anesthesia, which method has proved very satisfactory.

SHOCK.

The exact causation of so-called surgical shock which exhibits itself independently of hemorrhage is still under dispute. We can safely say that in shock we are probably dealing with either a reflex vaso-motor or cardio-inhibitory condition or a combination of the two. Shock may manifest itself as a very transient syncopal condition requiring no treatment or it may occur as a marked circulatory disturbance progressing to a fatal termination.

HEMORRHAGE.

Hemorrhage *per se* must be in considerable amount in the healthy individual to cause any marked circulatory disturbance. In very much weakened individuals or in those too freely purged a far smaller amount of hemorrhage will cause trouble. Today with the technic of the surgeon so near perfection hemorrhage is rarely the cause for circulatory disturbances.

PREVENTIVE MEASURES.

When considering the various factors causing circulatory disturbances it will be noticed that they

*Read before the Interstate Association of Anesthetists, Louisville, Ky., July 26, 1916.

may almost without exception be termed preventable causes. A careful anesthetist will certainly not err upon the side of over-dosage unless there be an idiosyncrasy on the part of the patient. Further, with chloroform anesthesia particularly and with nitrous oxid and ether to a lesser degree a small preliminary dose of morphin and atropin will lessen the tendency toward circulatory symptoms.

Asphyxia is a factor over which the anesthetist must exercise the greatest care. Even a slight degree of cyanosis should not be permitted for any length of time. Under all conditions oxygenation sufficient in degree must be constantly maintained.

In preventing surgical shock the surgeon must contribute his share, which consists in gentle manipulation of the tissues and viscera and in controlling hemorrhage. The anesthetist is often unjustly blamed for circulatory disturbances arising during anesthesia when in reality the surgeon has produced the condition by roughly handling the tissues.

If the patient has been greatly weakened by preoperative conditions such as too free purgation, constant vomiting or hemorrhage, saline, either subcutaneous or intravenous, should be given before the anesthetic is commenced. If the patient is particularly nervous it would perhaps be best to anesthetize first and then immediately begin the saline.

TREATMENT.

When circulatory failure occurs it is practically always secondary to respiratory failure and measures for relief should embody this fact. Given a case of circulatory failure, either partial or complete, the general treatment should be as follows:

(1) Cease the administration of the anesthetic.

(2) Invert the patient by the extreme Trendelenburg position, or in case of children, by grasping the limbs and swinging the head downward. Unless the signs of circulatory failure are very marked inversion of the patient is unnecessary and we need only to practice artificial respiration.

(3) See that an open air-way is present and begin the administration of oxygen. Artificial respiration should also be begun if the respiration has entirely ceased. The introduction of a Hewitt-Ferguson, or Connell air-way will usually insure sufficient breathing space without resorting to traction on the tongue. In the majority of cases the administration of oxygen and practice or artificial respiration is all that is necessary in treating this condition.

When chloroform has been the anesthetic ether may be used subcutaneously as a stimulant. When the circulatory phenomena have disappeared ether may then be substituted for the chloroform.

Strychnin is considered of no value by certain men and lauded by others. Personally I have undoubtedly seen good results follow its use and I should prefer to follow the teachings of Yandell Henderson and use strychnin.

Pituitrin has been recommended and probably does exert a beneficial effect. This has not been tried in a sufficiently large number of cases to determine its exact worth in anesthesia.

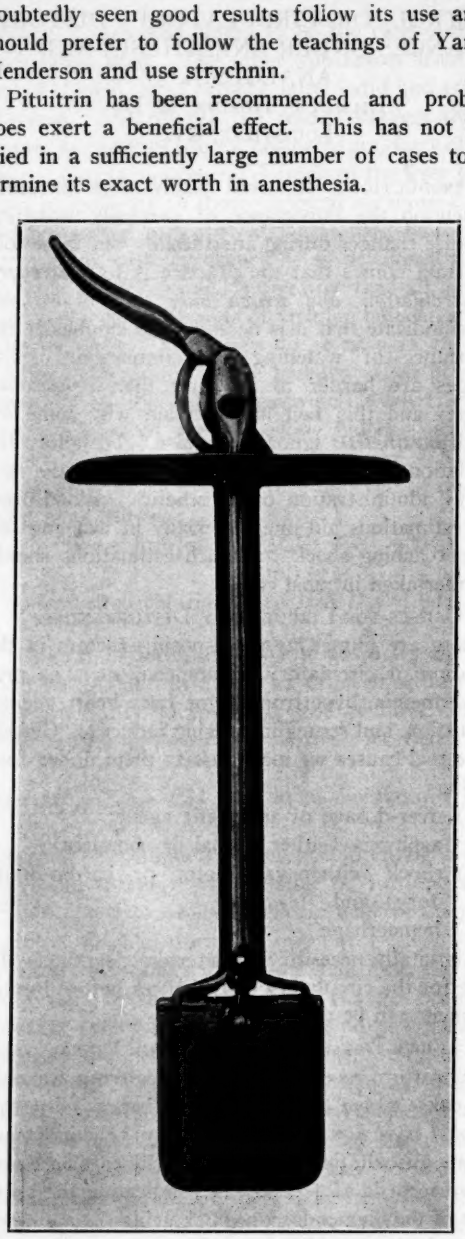

Fig. 1. Instrument for direct massage of the heart, closed for introduction.

In the past when chloroform was the anesthetic epinephrin was recommended as of great value, either subcutaneously or intravenously. The investigations of Levy seem to prove that epinephrin is particularly dangerous in chloroform anesthesia in that it produces auricullar fibillation, which is practically always fatal.

As already mentioned, either intravenous or subcutaneous saline is of no value unless there has been

actual loss of body fluids, either pre-operatively or during the procedure. The value of the Hogan gelatine solution must be determined later and I do not believe that one would be justified in administering such a solution at the present time.

It must be remembered that since the circulation is at fault the administration of drugs except in mild cases of circulatory disturbances is absolutely valueless.

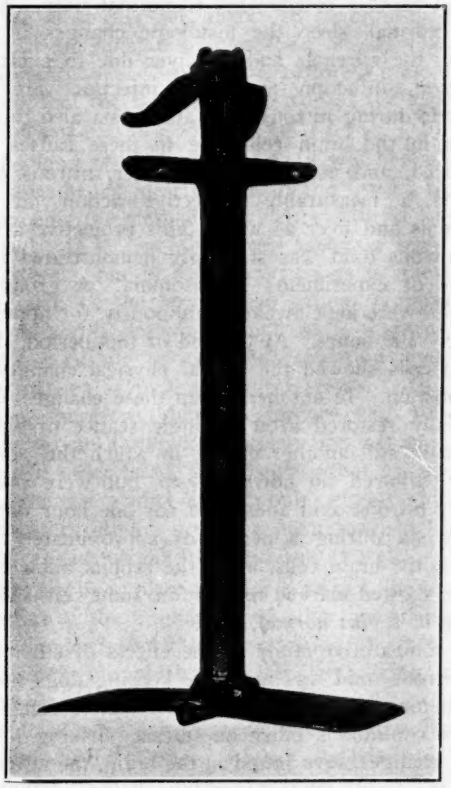

Fig. 2. Same instrument opened for use.

DIRECT MASSAGE OF THE HEART.

Should the measures already outlined fail to cause a return of the heart action after four or at the most, five minutes, direct massage of the heart should be practiced. For this purpose we have at our disposal three methods:

(1) Subdiaphragmatic massage of the heart.
(2) Transdiaphragmatic massage.
(3) Direct massage after opening the thorax.

Subdiaphragmatic massage is the easiest method to use, particularly if the operation is an abdominal one. The technic is simple and consists in manipulating the heart through the diaphragm and making rhythmic pressure about seventy times to the minute. There is no danger of pneumothorax which usually follows the thoracic and which also may follow the transdiaphragmatic method. Of the cases recorded in the literature the subdiaphragmatic procedure has been followed by the greatest degree of success.

The method of transdiaphragmatic massage necessitates also that the abdomen be open. The diaphragm is incised a little to the left of the median line and two or three fingers introduced through the opening. The fingers rhythmically press the heart from behind up against the sternum. This method, while apparently better than the subdiaphragmatic, has not been followed by success.

To fully expose the heart by opening the thorax would seem to be the best method, but it has several disadvantages. In the first place the opening must be carefully made and the pleura protected from injury as much as possible. This takes time and each second counts in such cases. Further, unless one is operating by some method which prevents pneumothorax this must be considered. On the whole this method has not been followed by as good results as the subdiaphragmatic, and it is more time-consuming and more likely to be followed by serious consequences. This method, so far as successful results are concerned, occupies a position midway between the subdiaphragmatic and transdiaphragmatic methods.

In addition to the three methods already mentioned for massage of the heart I desire to call your attention to a fourth one—that of instrumental massage. An instrument is employed which consists of two spade-like blades 27 x 25 mm. folded together. A lever controls the blades so that when open they present a flat surface measuring 54 x 25 mm. The total length of the instrument is 20 cm.

In using this instrument the method employed is extremely rapid and simple. A small area over the precordia is painted with iodin. With a knife an incision 3 cm. in length is made over the fifth interspace, slightly to the right of the nipple. This incision extends down through the skin, muscle and pleura hugging the upper border of the rib. The folded instrument is quickly introduced and then extended, thus bringing a flat surface into place for making rhythmic pressure on the heart. Experiments on dogs, which will soon be reported, would seem to indicate that this offers a feasible and very rapid method for massage of the heart. I should not advise the use of this instrument until subdiaphragmatic massage had failed.

Circulatory trouble arises usually when least expected. The anesthetist must therefore not only be prepared for such emergencies, but he must meet them without delay.

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AN EXPERIMENTAL RESEARCH INTO THE NATURE OF NITROUS OXID AND OF ETHER ANESTHESIA, WITH SPECIAL REFERENCE TO: (A) CERTAIN EFFECTS ON THE ORGANS OF THE BODY; (B) CERTAIN RELATIONS TO NORMAL SLEEP; (C) H-ION CONCENTRATION AND (D) INFECTIONS.*

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If we view man and the lower animals as adaptive mechanisms, governed by the universal laws of physics and chemistry, then we must believe that every phase of human or animal behavior is occasioned by and in turn occasions physical and chemical changes in the body. Therefore an understanding of the vast alterations in the behavior of the anesthetized organism—changes which may range from the slightest depression of mental power through progressively deeper stages of unconsciousness to death, necessitates a search for the physical and chemical changes caused by the anesthetic. Accordingly, in this study we shall consider (a) the gross behavior of the anesthetized individual in a consideration of the relation of anesthesia to normal sleep; (b) the influence of ether and of nitrous oxid on the histologic structure of certain organs; (c) the relation of ether and of nitrous oxid to the hydrogen-ion concentration of the blood and to reserve alkalinity; (d) the relation of ether and of nitrous oxid to the infections; and finally, (e) the clinical bearing of these studies.

(A) *The Relation of Inhalation Anesthesia to Normal Sleep.*—The outward similarity in the appearance of sleep and of anesthesia needs no comment. The manner in which a child becomes unconscious under a sufficiently concentrated flow of nitrous oxid over its face closely resembles the manner in which the healthy normal child falls asleep. That sleep is merely the depression of consciousness and that sleep only partially suspends the function of the brain is indicated by slight responses to stimuli, such as the shifting of posture; moving when called or when bright light strikes the eye; the response to tickling, or other reflexes. In other words, the receptors are still active during sleep. The activity of the receptors in anesthesia is evidenced as clearly by the increased pulse and respiration in response to trauma; and, if the anesthesia be light, by increased muscular tone, even by muscular movements. Another similarity is found in the resemblance between dreams during nitrous oxid anesthesia and the dreams of normal sleep.

(B) *The Influence of Ether and Nitrous Oxid on the Histologic Structure of Various Organs.*—As far as nitrous oxid is concerned, here also is found another resemblance to normal sleep. During normal sleep the histologic changes in the brain, the adrenals and the liver due to exertion, emotion, infection and acid injection, are repaired; during nitrous oxid anesthesia also the lesions in the brain cells due to these causes are repaired, and as will be seen later, nitrous oxid exerts a measurably protective action on the adrenals and liver as well. This protective action of nitrous oxid was strikingly demonstrated in a series of experiments on insomnia. A group of rabbits were kept awake continuously for approximately 100 hours. At the end of this period, their brain cells showed the typical physical changes of exhaustion. In another group these changes were partially restored after a single seance of sleep; while in still another group, in which the rabbits were allowed no normal sleep, but were placed under nitrous oxid anesthesia for one hour out of every six during a period of approximately 100 hours, the brain cells, as in the rabbits allowed to sleep, showed marked restoration, some cells indeed being in a *plus normal state.*

A comparative study of the effects of ether and of nitrous oxid was made by keeping dogs under continuous anesthesia for four hours. After four hours continuous ether anesthesia, striking histologic changes were found in the brain, the adrenals and the liver; while after four hours continuous nitrous oxid anesthesia, there were practically no changes in the brain and but slight changes in the liver and adrenals.

(C) *The Relation of Ether and of Nitrous Oxid to the H-ion Concentration of the Blood and to Preserve Alkalinity.*—In a series of H-ion concentration tests made for me by Dr. M. L. Menten, it was found that the H-ion concentration of the blood was increased by ether and by nitrous oxid: that the acidity increased more rapidly under nitrous oxid than under ether, but that the total acidity in either case was proportional to the depth of anesthesia.

In measurements of the reserve alkalinity of the blood made for me by Dr. W. B. Rogers, he found that during anesthesia the reserve alkalinity gradually decreased, the total decrease being greater under ether than under nitrous oxid. He found

* Read during the Fourth Annual Meeting of the American Association of Anesthetists, Detroit, Mich., June 12, 1916.

also that the alkalinity of the spinal fluid and of the bile was altered during anesthesia.

It is significant to note here the fact that the histologic lesions in the brain, the adrenals and the liver, caused by ether and by nitrous oxid, though varying in degree, are identical in kind with those caused by the intravenous injection of acids, as with those resulting from insomnia, exertion, emotion, infection or physical injury.

We must conclude, therefore, that the histologic lesions of inhalation anesthesia are acid lesions; and since acid lesions are restored only during sleep, that the same must be true of the lesions of anesthesia.

As long as fifteen years ago, it was believed that all anesthetics interfered with the use of oxygen, this being the method by which anesthesia was produced. It is true that deep asphyxia may cause anesthesia, but in such asphyxia as is due to obstruction of the larynx, the patient remains conscious and feels pain, though he is markedly cyanotic. While under good inhalation anesthesia, the patient is pink and unconscious. It would seem, therefore, that interference with oxidation alone is an insufficient explanation of anesthesia. On the other hand, the postulate that anesthesia is an induced acidity is not only supported by the laboratory studies we have mentioned, but by the clinical phenomena of anesthesia as well.

It may be in place to refer here to Lillie's notable studies of the mechanism of ether anesthesia. He has demonstrated that the addition of ether to the seawater in which arencola are living caused changes in the semi-permeable membranes, as a result of which these membranes become less permeable to the passage of ions. As life itself is dependent upon the permeability of the cells of the organism to the passage of ions, this action of ether suggests a further explanation of anesthesia, though its application has not been worked out in detail.

(D) *The Relation of Ether and of Nitrous Oxid to the Infections.*—We have already referred to the protective action of nitrous oxid in the presence of the stimuli of continued consciousness as in prolonged insomnia; and other histologic studies have shown that morphin also protects the brain, adrenals and liver from the destructive action of prolonged or intense stimuli. It follows, then, that since the effects of an infection upon the brain, adrenals and liver are identical with the effects of insomnia and emotion, then the administration of nitrous oxid and morphia will in part protect the organism against the damaging effect of infection.

Our experimental findings may be summarized as follows: Nitrous oxid and ether anesthesia alike cause an increase in the H-ion concentration of the blood, the spinal fluid and the bile; and progressively decrease the reserve alkalinity of the blood. Ether causes marked histologic changes in the brain, adrenals and liver, these changes being identical in kind with the histologic lesions caused by acid injection, or activation of any kind. Nitrous oxid and morphia each measurably protect the brain against histologic changes due to infection, while ether increases the damaging effects of infection.

(E) *The Clinical Bearing of These Studies.*—The identity of the phenomena of anesthesia with the succession of clinical symptoms which accompanies an increasing acidosis combined with the fact that the histologic changes produced by nitrous oxid and by ether are identical with those caused by acidosis, supports the postulate that anesthesia is an induced acidity. If the acidity is slight, anesthesia is light and the patient responds to even slight stimulation. As the acidty increases, the anesthesia deepens—first, associative memory is lost, but the cutting of the skin still causes involuntary movements; then muscular tone is lost and even the strong contact stimuli of a surgical operation cannot drive their impulses through the brain to the muscles; and finally, the decreasing alkalinity may so nearly approach the neutral point that even the circulatory and respiratory centers, which are especially adapted to respond to the stimulus of increased H-ion concentration, fail to respond; respiration and circulation are suspended and acid death—*anesthetic death* follows.

These studies explain why a patient, whose reserve alkalinity has been seriously reduced by exertion, emotion or physical injury, or infection; or by reason of starvation, interference with respiration or impairment of the liver or kidneys, is approaching acidosis, and does not take an inhalation anesthetic well; why there is much nausea and slow recovery from anesthesia in some cases and death in others; why children in particular, who are near acidosis, always pass into that state and die unexpectedly.

These facts show how necessary it is for the surgeon and the anesthetist alike to realize that during the operation each is draining the store of reserve alkalinity. If the surgeon employs a local anesthetic, uses gentle manipulation and produces the least possible trauma, he conserves his patient and demands from the anesthetist less of the damaging inhalation anesthetic. The anesthetist in turn conserves the patient by using the lightest pos-

sible even anesthesia administered with the least psychic trauma.

These studies show that for the bad risk nitrous oxid anesthesia is to be preferred to ether and that analgesia with local anesthesia should be employed, with general anesthesia only when it is demanded by certain phases of the operation.

These researches suggest the value of a mechanistic view of the phenomena of anesthesia and attach a high importance to the work of the anesthetist.

In my own clinic we have now administered nitrous oxid anesthesia in just under 15,000 cases without a death; and moreover, as our knowledge and experience accumulate, we are able with increasing accuracy to adapt the anesthetic to the individual.

1021 PROSPECT AVENUE.

A STUDY OF THE DISPUTED POINTS IN LOCAL ANESTHESIA.*

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In the discussion of this subject it is my aim to avoid predilections for and prejudices to any drug or method, permitting the drug or technic to stand on its own merits.

Much that I will say can only be verified or disproved in the clinic, the laboratory offering no, or very little opportunity towards a satisfactory solution of certain phases of local anesthesia.

Some time ago Dr. Leo Stern of New York published an article[1] in which he maintained that infiltration anesthesia with novocain suprarenin, resulted in hyperaemia and death of pulps. In another and later publication[2] he elaborates on his first observations and reaches the conclusion that "infiltration anesthesia should *never* be used in the oral cavity".

These are startling case records and necessarily result in a startling conclusion.

It would not be fair to myself if I didn't here, at the outset, reiterate[3] that I personally rarely employ infiltration in my work. And my sole purpose is to learn whether or not it can be employed with safety.

In order to freshen your memory with the controversy that has arisen between Dr. Stern and myself, permit me to briefly quote the substance thereof:

I criticized[3] his advocating the pumping motion—"moving the syringe backward and forward" when performing the maxillary tuberosity and mandibular injections, because this method tunnelled through the tissue unnecessarily and because[3] it would, by repeated jabs, do the things that those who practiced this method wished to avoid. To this, in answer to my criticism[4] he replies that Braun advocates this practice and then quotes Braun as follows:

"To avoid injecting large quantities of the anesthetic into a vein (and thus avoiding toxic effects) the syringe must be in constant motion, injecting during the insertion and withdrawal of the needle."

As is obvious from this quotation, Braun does not advocate pumping, and his fear of injecting directly into a vein is unwarranted, as applies to the dental regions and moreover, such accident is harmless, providing the solution is injected slowly. As a matter of information, I must add that it was Riethmüller who, in October, 1914, first called my attention to the adoption of the pumping motion, but has since concurred in my denunciation of the practice.

Let us now examine the most interesting part of contention, that is, whether or not to employ infiltration.

That thousands of operators throughout the world employ infiltration anesthesia without the above-mentioned sequelae, is a fact too obvious to mention. And I haven't either in print or correspondence been able to find other instances than those Dr. Stern mentions. Desiring, therefore, to test this point of contention in as near a laboratory manner as possible, I hit upon a plan that I think is certain to either commend or condemn it, but of this we shall speak later.

Dr. Stern is of the opinion that the "vaso-constrictor action of suprarenin" plus the "mechanical damage subsequent to the great pressure required to force the solution into deep structure" are to blame for deaths of pulps; to which I must say that by diffusion and not by pressure, does the solution pass through the bone, and as Dzierzawsky has shown, using colored solutions in subperiostial injections, it does not require long for the bone to be permeated, and, however delicate tissue may be, it still has recuperative powers and the action of suprarenin, as will be shown later, appears to have no detrimental effect.

But these again are merely processes of laboratory reasoning. Permit me then to acquaint you with the experiments started.

Realizing that the average clinic is not a suitable place for the results sought, because after infiltration it is rather uncertain as to whether the patient will return at stated intervals for the purpose of ascertaining the vitality of pulps, I asked the first

* Read during the Joint Session of the Interstate Association of Anesthetists and the National Dental Association, Louisville, Ky., July 26, 27, 1916.

year students of our college to volunteer for infiltration injections just for the experiment. I chose the first year students for an obvious reason, namely: I have recourse to them for at least three years for observation.

The injections were all made in the presence of class members (whom on this occasion I wish to sincerely thank) and the technic employed was as follows:

The vitality of the right upper lateral was ascertained and when found devitalized, the left upper lateral was chosen, and in no case did we have to leave these regions. The gum was iodinized and 1½ cc. of a 2 per cent novocain suprarenin solution was injected and the area massaged. In no case has there been a report of trouble, and upon opening of college I shall at intervals re-examine our cases for this report. These experiments are necessarily slow, but we must build on facts.

A very important and probable factor where death of pulps has occurred following infiltration anesthesia, is the injury caused by over-heating with stone or bur. Though the tooth under anesthesia be insensitive to grinding, it should not be subjected to prolonged friction. A stream of water should be directed into the cavity during excavation.

Nor can the fact be entirely overlooked that some teeth we are called upon to treat are, as far as the pulps are concerned, already in a semi-devitalized state, and would, without any sort of an injection, die.

We now come to the very interesting phase of my subject. What is the cause of partial collapse under novocain, the pallor, the trembling, and faintness? Various authors contribute to the causes, but I have never seen any one book or article that lists all of the causes that induce these results.

Thus Dr. Kurt Thoma is certainly in error when he imputes the tremor, pallor, and faintness to suprarenin. And the *T* tablet which he advocates will produce tremors similar to the *E* because it is the novocain and not the suprarenin that is to blame. The solution that we usually employ, is made by dissolving one *E* tablet in 1 cc. of Ringer, the result being a 2 per cent. novocain solution, and the suprarenin in 1 to 20,000 solution. Dr. Thoma would have suprarenin in 1 to 70,000 and therefore be of no practical value. When we realize that physicians daily use a 1 to 1,000 solution (15 drops of same) with impunity, excepting in the aged, it stands as a safe procedure. *Moreover, the most practical test shows that pure novocain or cocain, when applied to an open wound, causes this tremor to appear, yet when suprarenin 1 to 1,000 is applied, this effect is not to be noticed.*

I have injected pure novocain into my forearm subcutaneously with the typical novocain tremor, which, by the way, is preventable, but on injecting 2 cc. of suprarenin solution containing the same amount of the drug that is contained in 2 *E* tablets (viz.: .00010 of a gm.) this effect could not be produced. The reason for this is obvious, when novocain or any other local anaesthetic is injected into the system, part of it immediately enters the circulation and as it passes the cardiac and respiratory brain centers, it effects them just sufficiently to create this flutter, this disturbance.

And now, can this novocain tremor be prevented? Or are we to allow a fear to spread throughout the profession and curtail its use? Already there is spreading a belief that there exists a novocain idiosyncrasy. And there is as much truth to that as there is to the superstition that there exists an idiosyncrasy to cocain. I hope that no one in this intelligent body is of that opinion, neither as concerns novocain or cocain. Certainly no one here believes that when sloughing resulted after some careless pioneer injected a cocain solution that had stood so long on a dusty shelf and had become so mouldy that it seethed with infection. I repeat, no one would credit the drug in this event, nor is it necessary to spend very much time ascertaining the truth of this statement, when you realize that in all the literature you have never heard of a clean cocain preparation ever having effected the most sensitive of tissue—viz., the eye, the urethra. And while on the subject, let me say that were it not for the fact that cocain is habit-forming and can not be sterilized by boiling, it would make an excellent substitute for novocain, in spite of its toxicity. But here you must be able to differentiate between toxicity as refers to the central nervous system, and local destruction of tissue (sloughing). The latter, cocain is incapable of doing, and its toxic effect on the central nervous system can also be prevented by proper care.

I suppose it is needless to state before this audience that in order to obviate tremors, partial faints, pallor and so forth, we must inject very, very slowly, and must have the patient in as near a supine position as is convenient.

The term *slow* is a relative one. Some operators empty a two cc. syringe in the five seconds and think that it is a slow injection. Others double this time and think that it is an exceedingly slow injection. But if you would remember one thing of what I have said, please bear this in mind: *"Take sixty seconds by the clock to empty your syringe, and you will have no untoward experiences to relate."* Dosage matters but little; I have often used more

than a tube of *E* tablets in surgical operations on the jaws. Concentration is of more moment than dosage, and even concentration is of less importance than the slowness of injection. In this connection I wish to call your attention to the dangerous maxim promulgated by Seidel at the Muenster Congress where he remarks that "It makes no difference whether a certain dose is injected in weak or strong concentration, as long as the total dose of .2 gm. is not transgressed.[5]" This is certainly dangerous teaching, and even the novice is aware that no local anesthesic is so harmless as to be used irrespective of concentration. A five-grain tablet of Bichloride of Mercury is certainly less toxic in a gallon of water than it would be in an ounce of the same. And so with .2 gm. of novocain.

Position has a great deal to do with prevention of the above mentioned results, and the more erect the patient is sitting, the slower should the injection be executed.

When a patient is in a fainting condition, our first step is to place him in a supine position, so why not approach as near this position as is convenient and obviate chance? You have often heard the expression—"he or she *fell* fainting", but I do not remember hearing of a faint from any cause, while lying down. Attention to these details is productive of uneventful injections and the so-called idiosyncrasies are not to be observed.

FOURTH NATIONAL BANK BLDG.

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(5) Quoted from Thoma's "Oral Anesthesia."

RELATIVE VALUES OF SO-CALLED WARMED AND UNWARMED ETHER VAPOR.*

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In this paper an attempt will be made to summarize the evidence bearing on the use of warmed and unwarmed ether vapor in anesthesia. By unwarmed ether vapor is understood ether vapor which has not been passed through some specially devised heating apparatus before delivery to the patient.

The average temperature beneath the mask when ether is administered by the open drop method is about 32° C—that is, relatively near body temperature. We may therefore eliminate the necessity of any apparatus to warm ether given by the open drop method since the enclosed space in front of the mouth, provided by the cone, forms a natural warming chamber from which the air-ether mixture is delivered into the mouth at a temperature approximating that of the body. McCarthy and Davis have shown that the further increase in heat in such cases is gained by the time the mixture has entered the pharynx, so that the entire warming process is accomplished before the anesthetic enters the respiratory tract proper.

In considering the advisability of warming ether vapor when delivered by other methods, the available data will be presented from the standpoint of safety, efficiency and comfort.

RELATIVE TOXICITY: In 1906 there was published[2] the results of a series of experiments with chloroform vapor on cats from which it was concluded that unwarmed chloroform vapor was 2½ times as toxic as warmed vapor. Since then the same author has investigated the effects of warmed and unwarmed ether vapor and nitrous oxid gas on cats, rabbits and guinea pigs, confirming his belief in the excessive toxicity of unwarmed anesthetic vapors.[3] This conclusion, if correct, would make the use of warmed vapors imperative. On examination of the reports of these experiments, however, it appears that by *toxicity* is meant anesthetizing power; that the same organs are similarly affected by warmed and unwarmed vapor, but that the latter produces its effects more rapidly. Accordingly, if unwarmed vapor is 2½ times as potent as is warmed vapor the anesthetizing and lethal doses of the former should be two-fifths that of the latter. In other words, if anesthesia could be maintained for ten minutes with one ounce of ether in the form of unwarmed vapor it would be necessary to use 2½ ounces of ether during the same time if anesthesia were continued with warmed vapor. There are certain facts which lead us to question the validity of such an assertion. In the first place, certain advocates of the use of warmed vapor have stated that warming the vapor decreases the amount required to maintain anesthesia and that the vapor will act with increased rapidity in proportion to its warmth.[4] Secondly, in a series of experiments on dogs, in which warmed and unwarmed vapor was administered directly into the trachea, we were unable to detect any difference in the quantity of ether required by the one or the other method to induce or maintain anesthesia!

* Read during the Fourth Annual Meeting of the American Association of Anesthetists, Detroit, Mich., June 12, 1916.

Moreover, anesthetic vapors, regardless of the method of administration, reach the alveoli at body temperature; even the small amount which may be absorbed through the mucous membrane of the nose and throat must be at body temperature when it reaches the capillary walls. When one considers the area of the respiratory surfaces and the mass of the body and circulating blood on the one hand and the mass and volume of anesthetic vapor of low specific heat on the other, it is inconceivable that the anesthetic could go into solution in the blood without instantly attaining body temperature. Therefore, no matter how cold the vapor may be when it enters the mouth, it is at blood temperature when absorbed and carried to the medulla, so that it seems impossible that variations in temperature outside of the body could in any way modify toxicity.

Careful examination of the reports shows a possible source of experimental error which may suffice to explain the findings without assuming a decrease in the toxicity of the vapors when warmed. In his work the investigator used a modified Junker inhaler, which consisted of a rubber bulb by which air was pumped through an ether bottle into a face mask provided with valves. The valves were so arranged that on inspiration the only source of air was the ether bottle, while on expiration the air escaped directly from the mask. Connected directly to the face mask was a very delicate rubber bag which served as an indicator and safety reservoir to prevent negative pressure in the face mask. The object of the experiments was to determine the rate at which animals were anesthetized and killed; in order to be certain that all of the animals received approximately the same volume of anesthetic vapor the latter was pumped into the mask at a rate sufficient to keep the indicator bag two-thirds filled.[14] When the vapor was warmed it tended to expand and the indicator distended proportionately. As the degree of distension of the indicator bag was the criterion of the volume of vapor being administered, any distension beyond the standard would be compensated for by decreasing the rate at which the air was pumped through the ether. It would seem, therefore, that the animals which received unwarmed vapor were anesthetized and killed more quickly than those receiving the warmed vapor, because they were given, not a more toxic, but a more concentrated ether vapor.

In our experiments[1] (mentioned above) the vapor was administered at a rate sufficient to maintain complete surgical anesthesia. By subtracting the number of cubic centimeters of liquid ether remaining in the ether jar from the number there at the beginning of the experiment and dividing the result by the duration of the anesthesia in minutes, it was possible to calculate the average rate at which the anesthetic was required. Thus it was found that the amount of ether required to induce or to continue an already induced anesthesia did not vary when the vapor was delivered alternately unwarmed and warmed, to the same animals. After the first half hour anesthesia was maintained by an approximately 6 per cent ether vapor whether unwarmed or warmed.

We may conclude that there is no satisfactory theoretical or experimental evidence that anesthetic vapors are made less toxic by being warmed; that there is evidence which suggests that warming the vapors makes little difference in the amount required to induce or to maintain anesthesia.

PRODUCTION OF SHOCK: It has been stated that the energy used in warming ether vapor to body temperature is a considerable factor in the production of post-anesthetic shock. The sources of these deductions have been in the main clinical observations of post-anesthetic convalescence and observations on the fall of temperature during anesthesia.

POST-ANESTHETIC CONVALESCENCE: Clinical observations are necessarily inaccurate because of the accompanying operative procedures and because of inability to properly and uniformly control other sources of heat radiation and use of energy. Some observers have stated that patients anesthetized with unwarmed vapor come from the operating room pallid, drenched in perspiration, vomiting and often in a state bordering on collapse, and imply that such conditions are attributable solely to the refrigerating effect of the anesthetic. In contrast to this is the opinion of observers such as Dr. Isabella Herb, who in a personal experience covering over 30,000 cases anesthetized with an open mask or by unwarmed vapor administered intra-pharyngrally, has found no such alarming conditions due to a well-administered anesthetic per se, even after prolonged anesthesia.[5]

HEAT LOSS: There is no doubt but that during anesthesia there is a progressive fall in body temperature, nor is there any doubt that the process of warming cold vapor to body temperature abstracts energy from the body. That the total fall in temperature is due to this use of energy is obviously not the case, for the factors of skin radiation and activity of sweat glands are of definitely greater importance in the dissipation of body heat. The question arises as to whether prevention of loss of heat by way of the respiratory tract is necessary or desirable. It has been demonstrated[1] that the coldest ether-vapor-air mixture which is ever likely to

be used for anesthetic purposes reaches a temperature of 8.5° C. and that the amount of heat required to raise to 37.5° C., a quantity of such vapor sufficient for an hour's anesthesia is approximately 3.95 Cal. The amount of energy expended in breathing air in an operating room at 27° C. (80° F.) is 1.15 Cal. per hour when no anesthetic is used, so that the increased energy required to breathe ether-vapor-air mixture at its minimum temperature is about 2.80 Cal. per hour. When passed through rubber tubing in a warm operating room the temperature of the vapor as it reaches the patient is considerably higher than 8.5° (at room temperature—Herb) and 1.0 to 1.5 Cal. will usually represent the heat expended in actual practice. Such a heat loss in a 150-lb. man could hardly be detected on a clinical thermometer. Any considerable drop in body temperature must therefore be due to other factors. Davis[6] published the results of clinical observations which seemed to indicate that the use of unwarmed ether vapor was responsible for an average heat loss which would be equivalent to 27.3 Cal. in patients weighing 150 lbs. This is a loss far in excess of what could be accounted for by the use of the coldest vapor one is likely to obtain from any vaporizing machine, so that it would seem that other factors must be responsible for the greater part of this decrease.

In an as yet uncompleted series of observations on dogs the authors have noted that, in a general way, there occurs a tremendous variation in the range of temperature in dogs of the same weight anesthetized under the same conditions, either by the open drop method or with unwarmed or with warmed ether vapor, and have also noted that the fall in temperature in dogs which have been subjected to abdominal section is greater than in those upon whom extra abdominal operations have been performed.

Dr. Isabella C. Herb and Dr. Frances Haines have furnished us with the following data gained from observations on a series of thirty-five patients anesthetized by the open drop method:

Number of patients—
(a) males = 19
(b) females = 16
Total 35.

Average duration of administration of anesthetic—
(a) males = 35 min.
(b) females = 35 min.
35 min.

Average fall in temperature—(rectal)—
(a) males = .36° C.
(b) females = .15° C.
.26° C.

Average cone temperature—
(a) males = 31.35° C.
(b) females = 32.2° C.
31.9° C.

Average room temperature 27.0° C.
Average amount of liquid ether 154.2 cc.
(a) males 183.0 cc
(b) females 120.0 cc

Through the kindness of Miss Emma M. Rohtge, head nurse of the children's department at the Presbyterian Hospital, we are enabled to present the results of a few observations on the changes in body temperature during natural sleep. Observations were made only on children who were without fever and who were otherwise without abnormalities which might be expected to cause variations in temperature. The rectal temperature was taken in the evening in each case just after the child had fallen asleep and again after an interval of two hours without waking the child. In eight of the children, two or three such observations were made in the course of a week; in two there was but one such observation.

Number of children—
(a) male = 5
(b) female = 5
.10

Average age of children—
(a) male 9.6 years
(b) female 7.2 years
8.4 years.

Average fall in rectal temperature during two hours of natural sleep—
(a) male .9° F.= .5° C.
(b) female .42° F.=.23° C.
.66° F.=.44° C.

In comparing these two sets of observations two very noticeable similarities appear. First, in each, there is a definite decrease in body temperature following the onset of unconsciousness; second, in each, the decrease in the temperature of the males is more than double that of the females. The fall in the temperature of the children comfortably asleep in bed for two hours is nearly twice that of the patients anesthetized with ether for 35 minutes.

Without entering into an exhaustive exposition of the subject, the evidence suggests that the decrease in body temperature due to etherization is strictly analogous to that occurring in natural sleep; but that it is a purely physiological process, and as such, cannot be regarded as evidence of pathological changes in the tissues.

A difference between natural sleep and sleep due to an anesthetic is that, as a general rule, if the body temperature approaches pathological extremes during natural sleep, the body is irritated even to the point of wakefulness, until protection is obtained, while during an anesthetic sleep, especially deep surgical anesthesia, there is no such mechanism for self-protection, and a dangerous fall in temperature may ensue if the body is not protected by sufficient covering. This does not mean that every avenue for the escape of heat must be

shut off, for heat production continues, though at a decreased rate, and hyperthermia with its exhausting influence may be induced.

There have been two deaths attributed to hyperthermia where warmed ether vapor was administered.[3] In each case there had been an operation involving the central nervous system so that the causal relationship of the anesthetic could not be determined, but it seems well to bear in mind that such an occurrence is possible. An anesthetized patient, well covered, will frequently maintain a uniform temperature. If now the only remaining avenue for the escape of heat is closed, and the patient loses the unquestioned stimulating effect of inhaling cool air, hyperthermia may occur, just as insolation occurs to some individuals on oppressive, muggy days in summer when the temperature of the air stays around 38° C. Instances have come to our attention where patients have complained greatly during the induction of anesthesia with warmed vapor because of its oppressiveness and have expressed great relief when the anesthetic was changed to an unwarmed vapor.

Taking the fall in temperature during comfortable, natural sleep as a standard, we have yet to see statistics demonstrating decreases in temperature, said to be due to the use of unwarmed anesthetic vapors, which approach the pathological.

Note has been made of the difference in the degree of the fall in temperature between male and female in the above observations. This, we know, is nothing more than a coincidence; nevertheless, we suggest that we may have here the key to the reported differences in the degree of the fall of body temperature in patients anesthetized with previously warmed ether vapor and in patients anesthetized with ether vapor which has not been so treated.

LOCAL IRRITATION AND CHEST COMPLICATIONS.

This is a subject which is rather difficult to treat satisfactorily because the available data, especially from the clinical side, consists of opinions and not of material which lends itself to critical analysis. The advocates of warmed vapor appear to regard it as a perfect prophylactic in all respiratory disorders incident to inhalation anesthesia. Cough and laryngeal spasm is hardly ever seen.[7] "Dryness of the throat is never complained of unless oxygen has been added." "Cold produces the so-called irritation of ether, contributing to nausea, vomiting and shock, delays the return to nutrition, disturbing the stomach by ingestion of ether-laden secretions, annoys the patient by leaving a lingering after taste on the breath, due to impairment of the eliminative functions of the mucous membranes."[4] In contrast to this, we again call attention to the testimony of Dr. Isabella Herb, who, in her large personal experience, and in a careful study of the work of fellow anesthetists, has failed to find any such disparity as that outlined above.

Graves[8] reported a series of 4,000 consecutive extensive operations under ether administered mostly with a paper cone, the end of which was covered with a few layers of gauze to permit of free passage of air. This series showed 37 chest complications of all sorts, with 2 deaths, a mortality of .05 per cent. and a morbidity of .92 per cent. Risley[9] reported a series of 1,920 similar cases with 27 chest complications and 2 deaths, a morbidity of 1.4 per cent. and a mortality of .1 per cent. In each of these two series 3 cases gave definite physical signs of pulmonary tuberculosis. Munroe,[10] using the open drop method, reported 1,000 cases with 15 chest complications and 2 deaths, a morbidity of 1.5 per cent., mortality of .2 per cent. In Graves series the anesthetic was administered by students under expert supervision; in the other two series by internes. Graves further analyzed a series of 2,000 cases and found that chest complications occurred after laparotomy 10 times as often as after extra abdominal operations of the same duration, and much less commonly after pelvic operations alone than after operations higher in the abdomen. In 920 extra abdominal operations he had only 2 chest complications, one of these in a septic case, a morbidity of .21 per cent. Kronlein[11] reported 1,409 laparotomies etherized by a trained anesthetist by the open drop method, a mortality from chest complications of 0.56 per cent. Gottstein[15] found more respiratory complications following operations under local anesthesia than he did following equally extensive operations under chloroform.

In view of the disproportion in the incidence of chest complications after laparotomies and after septic cases as compared to extra abdominal clean cases, and in view of the chest complications following operations under local anesthesia, it seems open to question whether so-called unwarmed ether, as such, can be made to assume responsibility for them.

The material available from the clinics of the advocates of warmed ether-vapor is not sufficient to permit comparison with the above, though Läwen[12] concluded that the use of warmed vapor does not prevent pneumonia.

Using unwarmed ether intratracheally on dogs, Meltzer[13] did not notice unusual evidences of irritation. McCarty and Davis[1] found no difference in the quantity of saliva and mucous secreted by dogs anesthetized with warmed and with unwarmed ether vapor, nor did they find appreciable

differences in the reaction of the respiratory mucous membranes as evidenced by changes visible to the naked eye, or by the course of the convalescence.

There is no proof that the preliminary warming of ether vapor decreases respiratory complications, or that failure to warm the vapor increases them; there is evidence that the procedure does not prevent post-anesthetic pneumonia.

SUMMARY AND CONCLUSION.

There is as yet no proof that warmed air-ether-vapor gives a greater margin of safety in anesthesia than the unwarmed vapor; it may be distinctly dangerous by aiding in the production of hyperthermia.

The assertion that preliminary warming of the vapor decreases pulmonary irritation is open to question.

The warmed vapors do not always add to the comfort of the patient, they may be distinctly uncomfortable.

Gram for gram of ether, there is no difference in the efficiency or toxicity of warmed and unwarmed vapor.

From the standpoint of simplicity and general adaptability, the unwarmed vapor is superior.

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ANESTHETIC RECORDS AND STATISTICS OF ANESTHESIA.*

ALBERT H. MILLER, M. D.,
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"Since all phenomena which have been sufficiently examined are found to take place with regularity, each having certain fixed conditions, positive and negative, on the occurrence of which it invariably happens, mankind have been able to ascertain the conditions of the occurrence of many phenomena; and the progress of science mainly consists in ascertaining these conditions."—J. S. Mill.

IMPORTANCE OF THE SUBJECT.

An accurate system of records is the foundation upon which any scientific work must rest. While some medical problems may be solved by personal judgment, many questions can be answered only from statistics based on careful records. It has frequently happened that one who has an experience of thousands of cases with one anesthetic and who considers this agent free from danger and ideal in every way; as the result of a series of mishaps, suddenly undergoes a diametrical change of opinion. Judgment of the safety of an anesthetic resulting from a study of the statistics of many cases is not liable to such a change.

DIFFICULTIES ENCOUNTERED IN COLLECTING STATISTICS OF ANESTHESIA.

To collect accurate statistics of anesthetic mortality and post-anesthetic complications should be a simple matter. Such statistics should be obtained from vital statistics of communities, from hospital records, and from records of surgeons and anesthetists. Difficulties are encountered in obtaining statistics from each of these sources. It often happens that deaths from anesthetics are not reported as such in the vital statistics of communities. A search of such statistics for known anesthetic deaths finds them frequently falsely reported as due to cardiac failure, uremia, or to the condition for which the operation was indicated. Hospital records are generally so inefficient that the expense of collecting statistics of anesthesia from them is prohibitive. The attempt to collect statistics of the anesthesia in hospitals independently of the hospital record system has always failed. The records of individual anesthetists or surgeons are not usually so kept as to provide accurate statistics. A mass of anesthetic records untabulated or not readily tabulated is of little value. The manner of administrations of any agent can be learned but the percentage of deaths and of post-anesthetic complications could be obtained only at a prohibitive expense. It follows that our knowledge of the re-

* Read during the Fourth Annual Meeting of the American Association of Anesthetists, Detroit, Mich., June 12, 1916.

sults of anesthesia consists of opinions and not facts.

Statistics of Anesthesia Which Have Been Published.

In spite of these difficulties, statistics of anesthesia which are worthy of study have been published. The most valuable of these have been collected by observers who have reported series of cases and results coming within their personal knowledge.

Neuber[1] collected 71,052 cases of anesthesia with 24 deaths. In 20,613 chloroform cases the mortality was 1 in 2,060; 11,859 ether cases showed a mortality of 1 in 5,930; and 10,230 cases where ether was preceeded by chloroform, a mortality of 1 in 3,410.

Hewitt[2] collected 918,578 cases of chloroform anesthesia with a death rate of 1 in 3,082; and 443,900 ether cases with a mortality of 1 in 14,773.

Gwathmey's[3] notable collection of American Statistics comprises 448,866 cases with 111 deaths. 22,513 chloroform administrations showed a mortality of 1 in 1,608; 294,653 ether cases, 1 in 4,533; while among 14,878 cases of local anesthesia there was no fatality.

Peterka[4] reported 100,971 ethyl chlorid cases with a mortality of 1 in 11,219; McCardie[5], 12,000 cases with a mortality of 1 in 3,000; Hornabrook[6], 18,813 cases with no death; and Ware[7], 15,000 cases without a fatality.

Jones[8] reported 13,000 cases of nitrous oxid oxygen anesthesia with 1 death; Teter[9], 23,952 cases with 1 death; and Crile and Lower[10], 34,964 cases with no fatality.

Teter[11] also collected 934,924 nitrous oxid administrations, 32,172 cases of nitrous oxid and air, and 190,724 nitrous oxid oxygen administrations; a total of 1,161,820 cases with but 2 deaths.

Strauss[12] reported 22,717 cases of spinal anesthesia with a death rate of 1 in 494; Chiene, 12,000 cases with a mortality of 1 in 545; and Violet and Fisher[13], 30,000 cases with a mortality of 1 in 866.

Wood[14] reported 1988 Scopolamin cases with a mortality of 1 in 221.

The writer[15] has collected from sources which seem to be reliable, 2,171,461 cases of anesthesia by various agents and finds a mortality from ether of 1 in 8,010; from chloroform, 1 in 2,605; from ethyl chlorid, 1 in 13,365; from spinal anesthesia, 1 in 515; and from Scopolamin, 1 in 666.

Value of the Published Statistics.

While the published statistics do not show the absolute proportion of danger attending anesthesia, they indicate the relative safety of the different anesthetic agents. The statistics show the comparative safety attending the use of ether and nitrous oxid, and the greater danger with which spinal anesthesia and the use of scopolamin are accompanied. The value of the statistics is limited by lack of standards, especially in judgment as to what constitutes a death from an anesthetic. In one series of cases, the influence of the anesthetic in producing a death from shock on the operating table is not questioned, while in another series, a fatality due to suffocation from inspired vomitus is included among the anesthetic deaths. Statistics do not justify the opinion of the Committee on Anesthesia of the American Medical Association; that the use of chloroform should cease, except as a preliminary to ether. For an operation on the pharynx, requiring the use of the cautery, chloroform is probably the safest and most efficient anesthetic that could be chosen. The authority of this committee places the anesthetist who uses chloroform in such a case under a serious handicap. On the other hand, the statistics have proved that the greatest danger from chloroform is in the early stages of its administration and that the use of chloroform preliminary to a safer anesthetic is unjustifiable. In fact, Flemming, after analyzing 700 fatalities under anesthetics which had occurred in Great Britain, advocates ether for induction, even where chloroform must follow.

Duty of the American Association of Anesthetics to Collect Statistics of Anesthesia.

There is no doubt that the American Association of Anesthetists can in a few years collect statistics of anesthesia of the greatest value. A committee on statistics should be appointed by the association. Statistics should be reported to this committee by members of the association from their own knowledge or observation. In each series, the total number of cases, with the nature of the operations included, and the number of deaths, the cause of death being stated in each case, should be indicated.

Standard Anesthetic Chart.

The adoption of a standard anesthetic chart by this association will be an important step toward the collection of statistics of anesthesia. The chart should be simple, with the data arranged in the order in which they would naturally be set down. A loose leaf system will be found more convenient than a bound book or a card system. After consultation with a number of members of the association the following chart is recommended. The front of the sheet contains the data in Fig. 1.

On the reverse side of the sheet is a chart for recording by figures, data noted during the anesthesia. The graphic chart in general use is less efficient than

the form recommended, the time required to enter data on the graphic chart being about three times that required with the chart recommended.

The data of the chart on the reverse side of the anesthesia sheet are entered as follows:

Hour refers to hour of the day and not to the time of anesthesia.

Note in the first blank column:
Time anesthesia started by X.
Time anesthesia established by Y.
Time operation started by O.
Time operation completed by C.
Time recovery commenced by R.

Note in the column designated:
Pulse rate by figures.
Respiration rate by figures.
Pupil, approximate size, 0°.
Color,
Normal by N.
Pale by P.
Cyanotic by C.

Note under *Remarks*:
Blood pressure.
Rash.
Mucus, if excessive.
Stimulation.
Position,
Dorsal by D.
Erect by E.
Trendelenburg by T.
Lithotomy by L.

Features of the operation affecting the anesthesia.

Anesthetists who wish to keep more detailed records than are provided for in this simple chart will find a larger sheet following the plan of the simple sheet, convenient.

The data on the front of this sheet may be arranged as in Fig. 3.

On the reverse side of this sheet is a chart similar to the one illustrated but expanded to provide for entering the systolic and diastolic blood pressure and such other data as the anesthetist wishes to record.

In hospitals having an efficient record system, it is recommended that no independent records of anesthesia be made but that the results of anesthesia be obtained from the hospital record system. Where there is no efficient system of records, the system recommended for private work may be employed.

When the anesthetist employs a card ledger sys-

Anesthesia No.	
Anesthetic	
Date	
Time	A. M. P. M.
Place	
Surgeon	
Diagnosis	
Patient	
Responsible party	
Address	
Charges	
Payment	
Age	
Previous anesthesia	
False teeth	
Heart	
Lungs	
Urine	
Medication	
Operation	
Assistant	
Recovery	
Remarks	

Figure 1. Simple Anesthesia Chart Recommended.

Hour A.M. P.M.	Pulse	Resp.	Pupil	Color	REMARKS
5					
10					
15					
20					
25					
30					
35					
40					
45					
50					
55					
5					
10					
15					
20					
25					
30					
35					
40					
45					
50					
55					
5					
10					
15					
20					
25					
30					
35					
40					
45					
50					
55					

Figure 2. Reverse Side of Anesthesia Chart.

tem, the ledger cards may be filed alphabetically and furnish an alphabetical index of his cases.

Tabulation of Reports.

The results of each 100 cases should be tabulated, the tabulation including the nature of the operations, the anesthetics used, the post-operative complications, and the deaths, the cause of death in each case being indicated. The most efficient report consists of a tabulation of each 100 cases of anesthesia with a single agent for a single class of operations.

No.	Date	
Anesthetic	Method	
Time	A.M. P.M.	Place
Surgeon		
Diagnosis		
Patient		
Responsible party		
Address	Charges	
Date of examination		
Age	Appearance	
Previous anesthesia		
Dyspnea	Edema	False teeth
Heart	Lungs	
B. P. Sys.	Dias.	
Urine		
Medication		
Operation		
Assistants		
Nurse		
Consciousness returned		
Vomiting, Immediate		
Conscious		
Excessive		
Complications		
Remarks		

Fig. 3.

The results of the tabulation read directly in percentages. A tabulation of any multiple of 100 cases may be readily made from the original tabulations. Tabulation sheets are quadrille ruled. At the left of the sheet are the numbers from 1 to 100 and at the top of the sheet are indicated the data to be tabulated: for example;

Column 1 Case numbers.
Column 2 Operations, Designate.
Appendix by A.
Breast amputation by B.
Gall bladder operation by G.
Minor operation by M., etc.
Column 3 Handicapped patient designated by X
4 Designate Nitrous Oxide by X
5 Designate Oxygen by X
6 Designate Ether by X
7 Designate Chloroform by X
8 Designate local anesthesia by X
9 Preliminary medication by X
10 Excessive vomiting by X
11 Operative complications by X
12 Circulatory complications by X
Examples: Embolism, Phlebitis.
13 Respiratory complications by X
14 Urinary complications by X
Examples: Nephritis, Glycosuria, Acetonuria.
15
16
17 Death by X
18 Anesthetic death by X

Tabulation sheets should be furnished by the association and as many filled sheets as possible returned by each member to the committee on statistics.

Standards to be Established.

Certain standards should be determined by this association: for example; what constitutes a surgical operation, whether dental extractions, the application of plaster casts, manipulations done without anesthesia, and dressings done under an anesthetic should be included. It is suggested that a surgical operation shall be considered to be a procedure for which an anesthetic is administered or omitted for cause. A standard should be set for what constitutes a death from an anesthetic. An anesthetic fatality should be suspected when death occurs during the period before consciousness would be expected to return, when a delayed death of the type known to be characteristic of the anesthetic agent occurs, or when a fatality results from the failure of an organ not known to be diseased before the anesthetic was administered.

Intrinsic Value of Anesthetic Records.

The first advantage appreciated by an anesthetist as by an institution which keeps careful records of the results of anesthesia, will be a knowledge of the character of his own work. The ignorance of anesthetists in this respect is not more remarkable than the ignorance of the medical and surgical profession in regard to the results of medical and surgical treatment. Surgeons do not certainly know whether the routine use of the Fowler position has proved advantageous. Neither surgeon nor anesthetist can enlighten a patient who approaches a gall bladder operation as to his chance for developing some pulmonary complication. One states that his anesthesia is perfectly satisfactory, while patients and visitors to the clinic frequently decide that it is not satisfactory at all. One states that by administering oxygen to patients as they recover from the anesthetic he has abolished nausea and

vomiting, but nurses who are familiar with the after condition of the patients are frank in saying that the use of oxygen has had no effect in reducing nausea and vomiting. Accurate knowledge of present conditions in anesthesia as in medicine and surgery will lead to greater efficiency in the future.

37 EVERETT AVE.

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PRACTICAL METHODS OF ANESTHESIA.*

DR. JAMES T. GWATHMEY,
NEW YORK CITY.

Gentlemen, this is the second time I have had the pleasure of coming before this Association. I remember some years ago the satisfaction and encouragement I received from reading a paper before this body.

The word "practical" in this paper means:

(1) The anesthetic should be suited to the patient and should be modified, or changed entirely, as indicated.

(2) The introduction should be pleasant to the patient, with respiration, blood pressure and color reflex continuing normal.

(3) The maintenance of anesthesia should vary with the surgeon's needs; at the same time the patient's condition should be as nearly normal as possible.

(4) The transition from the anesthetic stage should be as gradual and as smooth as the induction, the patient emerging as from a quiet sleep, without pain, nausea, or conscious or unconscious vomiting.

All of this is accomplished by modern methods, not yet in common use by the excessively utilitarian surgeon employing one method and a nurse anesthetist.

We maintain that *Anesthesia* is a vital part of the practice of medicine, and that the adoption of any one method or anesthetic, regardless of conditions, is contrary to the fundamental principles that should govern all surgical operations and the administration of all drugs. It is most unfortunate for humanity that prominent surgeons have by their *practical* methods decided that anesthesia is *not* a part of the practice of medicine.

When a surgeon assumes responsibility for the narcosis he becomes an *Anesthetist*. It is almost impossible for a surgeon to keep up with the literature of his own specialty. That he is not up to date on this subject is proven by the type of anesthesia witnessed in some of the best known clinics of to-day.

It is begging the question to say that the nurse anesthetist is superior to the average interne. A physician specializing in anesthesia should have control of this department in every large hospital, in every medical college and in every large teaching clinic. It is inexcusable to have nurses in absolute charge of the anesthesia in large teaching clinics.

The nurse anesthetist has proven to be entirely *practical*, but we take it that this Association wants anesthesia discussed under the higher meaning of the word *practical*, and this we propose to do briefly.

PREPARATION OF PATIENT.

In addition to the premiminary narcotic medication to be described the patient should have sodium bicarbonate and lactose, of each one drachm every four hours, for at least 48 hours before the operation. If the urine is already acid, the dose of sodium should be increased; glucose per rectum should also be given after the operation (Buckler).

Bonnot and Cleveland of St. Louis, after using quinin salts in 600 cases, recommend quinin muriate, grains ten, dissolved in two ounces of water at 100 degrees Fahrenheit, to be given immediately after operation; followed in 30 minutes by saline proctolysis or (in septic cases) by six ounces of olive oil. The quinin is repeated every six hours, for four to six doses. It is stated that with this medication *"bachache is practically eliminated and only about 2 per cent suffer gas pains."*

PRELIMINARY MEDICATION.

This should vary not only with the patient, but with the individual anesthetist. The consensus of opinion is that nearly every patient should have some form of preliminary medication; otherwise a rise of blood pressure at the very thought of the operation, together with other changes from the normal, prevents the smooth induction, the easy maintenance, and the uneventful recovery that we would otherwise obtain.

In preference to morphin, which sometimes in

* Read during the meeting of the Association of Eastern Railroad Surgeons, New York City, 1916.

itself produces nausea, we prefer the following preliminary, which is modified to suit the individual case:

Paraldehyde
Potassii bromide aa 1 to 2 drams
Aquæ qs. ad. 4 oz.

M. Sig. With patient in Syms position, give per rectum, 40 to 60 minutes before operation.

It has been proven in the laboratory that with this medication: (1) Anesthesia comes on sooner, and less ether is required. (2) The margin of safety is increased, thus making anesthesia a safer procedure.

This medication may possibly have the same effect as Crile seems to have found that morphin has,—*i. e.*, it regulates the acidosis; in fact Crile recommends bromides per rectum for this purpose. Codein may also be used instead of morphin.

Anesthetic Agents.

While morphin has long since been discarded as a general anesthetic, it is well to bear in mind, that in railway emergency work a patient is safer with morphin and alcohol than with either drug alone. Also, that with this combination an analgesic stage comes on before unconsciousness.

For instance, in the amputation of a limb, a tourniquet completes the analgesia long before the alcohol sleep commences.

Chloroform, anesthol and ethyl chlorid are rarely used in this climate as the terminal anesthetic; but as the initiatory agent, or to supplement other anesthetics, they are most useful.

The essence of orange (composed of 25 per cent oil of bitter orange peel, and 75 per cent absolute alcohol) is not in itself an anesthetic; yet its odor, being ten times as penetrating as that of other vapor makes it a safer and better introductory agent to ether by the closed method, than is nitrous oxid.

The physiological basis for the beneficial effect of the oil of orange in the administration of inhalation anesthetics may be found in the prevention of the reflex stimulation of certain sensory nerves by the anesthetic agent. Dastré attributed early syncope to reflex stimulation of the pneumogastric and trigeminal nerves, particularly the sensory branches supplying the nasal mucous membrane and the larynx. Embley has also emphasized the part played by the increased excitability of vagus mechanism, particularly during the early part of the administration. In his inhalation experiments Embley found that failure of respiration is mainly due to fall in blood-pressure. With good blood-pressure, failure of respiration (his experiments concerned chloroform) is practically impossible. Restoration of respiration is dependent upon restoration of blood-pressure. The chances of dangerous vagus inhibition are greatly increased by imperfect respiration.

From these findings it would seem fair to assume that any factor which prevents inhibition of the nervous mechanism of respiration, and which, by its stimulation of the respiratory centre, prevents fall in blood-pressure, will have a beneficial influence upon the course of the anesthesia. The oil of orange seems to exert this beneficial influence by dulling the sense of smell to such an extent that the odor of the anesthetic agent is not noticeable during the administration.

Essence of orange is also useful when ethyl chlorid, chloroform or anesthol are used as the introductory to ether by the open method.

When chloroform is given with the proper apparatus,—in which we have oxygen, air, and rebreathing throughout,—the patient goes under and comes out of the anesthesia as quickly as with nitrous oxid and oxygen and with immunity unimpaired.

In emergency practice when chloroform is used, it is safer and better in every way to surround the mask with paper or a towel and have rebreathing throughout, thus diminishing by one-half or less the amount of the anesthetic used.

Again, dipping the chloroform bottle in a vessel of hot water from time to time, also increases its safety.

Open Drop Ether is a crude, unscientific method of administering this drug. It is responsible for all the ill repute that ether bears to-day, especially as regards nausea and vomiting.

Administered by the best *artist* in the world it is irregular and uneven, with a tendency to acidosis and resultant shock on account of insufficient supply of oxygen.

The administration of nitrous oxid followed by ether, called the *gas-ether sequence,* is an even cruder and more dangerous method than the *open drop*. It raises the blood-pressure enormously, cyanoses the patient, and is responsible for many deaths.

In the light of our present-day knowledge there is no excuse for a continuance of these methods.

The nitrous oxid-oxygen ether sequence by the closed method removes all of the objectionable features of the *gas-ether* sequence, and should be used whenever a closed method is indicated.

The endo-tracheal method introduced by Meltzer has been called the *luxury of breathing,* but

the patient can be given this same luxury by the endo-pharyngeal method which is now used by those who formerly employed the Meltzer technic. The same results are secured, however, with the vapor mask which some of us have been using for over ten years.

The *vapor mask* is any chloroform mask covered with gauze and with some suitable arrangement for admitting the warmed oxygen ether vapor.

The ether may be vaporized by any air compressing device, or oxygen may be used from any ordinary low pressure oxygen tank; or the oxygen talk may be filled with air. These methods insure an even ether vapor tension.

If this vapor is passed to the mask just described, it is called open, or, more correctly, a semi-open

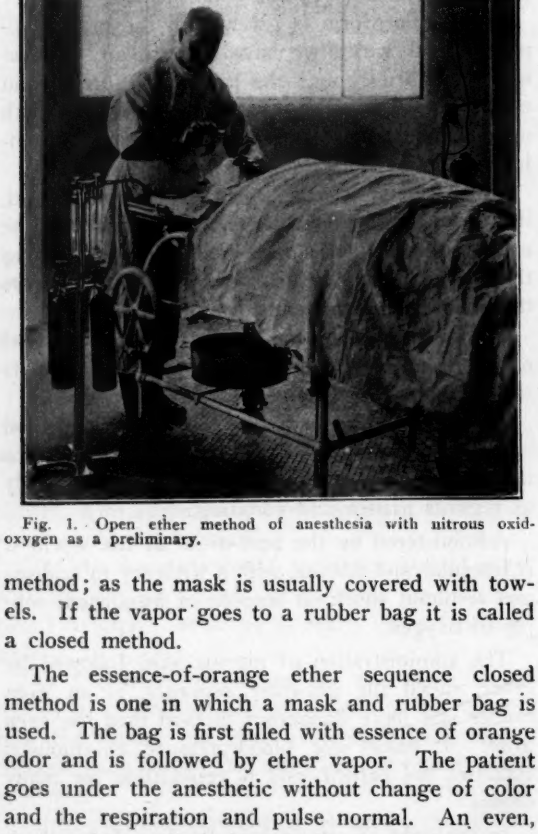

Fig. 1. Open ether method of anesthesia with nitrous oxid-oxygen as a preliminary.

method; as the mask is usually covered with towels. If the vapor goes to a rubber bag it is called a closed method.

The essence-of-orange ether sequence closed method is one in which a mask and rubber bag is used. The bag is first filled with essence of orange odor and is followed by ether vapor. The patient goes under the anesthetic without change of color and the respiration and pulse normal. An even, deep, safe anesthesia is maintained, and the patient comes out with as little discomfort as follows a good nitrous oxid-oxygen administration. Two and a half ounces per hour is the usual amount of ether used, as against four to eight ounces by the drop method.

The vapor mask, or semi-open method, should be used in operations on the head, neck and upper thorax; the closed method, in abdominal and other operations requiring relaxation.

The so-called gas-oxygen anesthesia supplemented with ether should be used in only selected cases and never as a routine. There is no method known to-day, either local or general, that should be used as a routine procedure.

Oil-ether colonic anesthesia is now a recognized method of administering ether. The patient falls asleep in his own bed, quietly, with pulse and respiration about normal, and the color reflex fully maintained at all times. This method is indicated wherever the element of fear is prominent,—as in

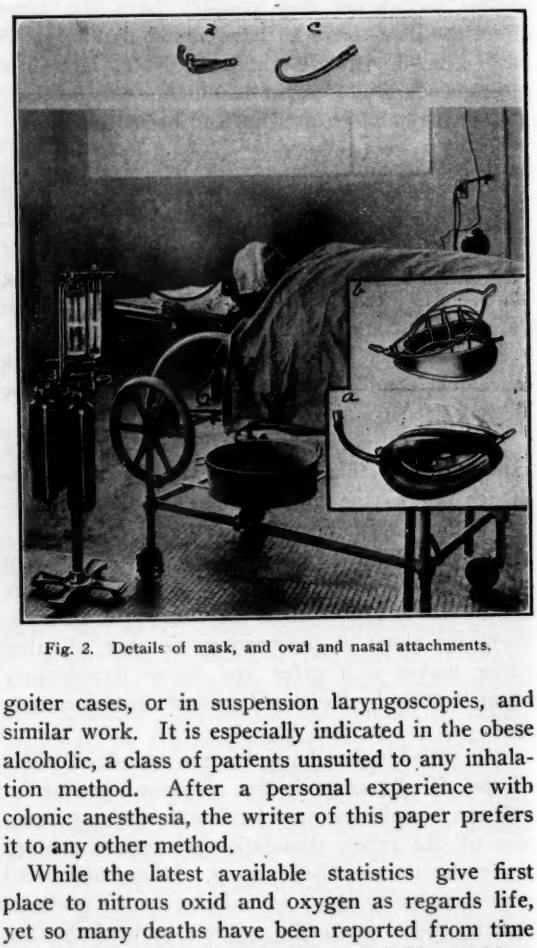

Fig. 2. Details of mask, and oval and nasal attachments.

goiter cases, or in suspension laryngoscopies, and similar work. It is especially indicated in the obese alcoholic, a class of patients unsuited to any inhalation method. After a personal experience with colonic anesthesia, the writer of this paper prefers it to any other method.

While the latest available statistics give first place to nitrous oxid and oxygen as regards life, yet so many deaths have been reported from time to time since the more general use of this combination, that we are forced to conclude that it is not so safe as is generally supposed,—that is, with our present methods of administration.

I have therefore devised, and am now using, a semi-open method of administering nitrous oxid-oxygen and warmed ether vapor. This ether-nitrous oxid anesthetic vapor is now warmed,

moistened, oxygenated, and deodorized. Administered in this way the patient does not at any time taste or smell the ether. The mask described is used, the patient getting at all times such an abundance of anesthetic vapors, that there is no strain whatever upon the respiratory center.

Unquestionably the preliminary and after treatment are quite equally as important as the anesthetic or the method of administration. The anesthetist should therefore have entire charge of this treatment, as well as the selection of the anesthetic, if he is to be held responsible for the condition of the patient after the operation. These conditions, granted, it can readily be appreciated that we make no rash promises to any patient in assuring him of a pleasant and safe voyage through the narcotic zone.

40 East 41st St.

THE EYE SIGNS OF NITROUS OXID ANESTHESIA.

A. H. O'Neal, A.M., M.D.,
St. Davids, Pa.

In giving nitrous oxid-oxygen anesthesia the observance of the eye is of equal importance to the pulse, respiration, blood pressure and color of the patient. Indeed, to me it has become a more reliable guide than any of these.

In watching the eye we note as the patient becomes unconscious, its continual oscillation, the contraction of the pupil and the lack of conjunctival anesthesia.

The next change to be noted is the *lack* of oscillation of the eyeball *with* conjunctival anesthesia. The eye muscles relax and the cornea is often seen in a dependent position. Here it is that the operation should be commenced. It is this loss of oscillation and conjunctival anesthesia (because the two are synchronous) which should thus guide us. If anesthesia is deepened the pupil will dilate. If anesthesia is lightened and the pupil still remains dilated and the eyeball oscillates, we have then a condition of shock which will show an oscillating eyeball with a dilated pupil. The operative work should then be temporarily suspended. Before asphyxia occurs to any great extent you will notice the oscillation of the eyeballs ceases, and I think it unquestionably gives us reliable information.

As the zone of operative anesthesia in nitrous oxid is but fleeting and narrow, I offer this sign of the non-oscillating anesthetic eyeball as the most reliable guide in its administration.

Society Proceedings.

Membership in a society devoted to your specialty is an essential to success.

1917 MEETINGS.

The American Association of Anesthetics will meet in New York City, June 2, just previous to the meeting of the American Medical Association. The Scientific Program and Dinner will be held in the Hotel McAlpin. The sessions will take place from 9 to 12 A. M. and 2 to 5 P. M., the banquet following immediately the same evening.

Among the papers already scheduled for the scientific program are the following: President's Address, Walter M. Boothby, Boston; Endopharyndeal vs. Endotracheal Anesthesia in Major Thoracic Surgery, Wm. Banower, New York City.. Ethyl Chloride Anesthesia, Martin M. Ware, New York City; Some Observations on the Scope and Usefulness of Narcoanesthesia, Henry Beates, Jr., Philadelphia; Comparative Studies in Blood Pressure, Albert H. Miller, Providence; Concerning the Effect of Anesthesia on the Blood Volume in its Relation to the Production of Shock, Albert A. Epstein, New York City; Some Observations on Ether-Oil Colonic Anesthesia, Arthur S. Brinkley, Richmond, Va.; Blood-Pressure and Graphic Vacomotor Changes in the Periphery During Ether Anesthesia, W. E. Muns, Columbia, Mo.; Demonstration of an Apparatus for the Determination of the Carbon Dioxid Content of Air and Blood, Yandell Henderson, New Haven, Conn.; and other papers by F. C. Mann, Mayo Clinic, Rochester, Minn.; Carl Henry Davis, Chicago; Charles H. Gallagher, Ithaca, N. Y.; with titles to be announced later.

The first two days of the meeting of the American Medical Association, June 4-5, will be devoted to clinics, during which arrangements have been made for the demonstration of the latest and most improved methods of anesthesia and analgesia. There will also be a Joint-Session with the Sections of Physiology and Pathology and General and Abdominal Surgery, during which several very important papers on anesthesia will be read, among them the following: Shock, F. C. Mann, Rochester; Pathological Pulmonary Changes during Endotracheal Anesthesia for Experimental Lung Surgery, Conrad George, Jr., Ann Arbor, Michigan; Further Observations on the Teaching of Anesthesia in Relation to Surgical Physiology and Pathology, Willis D. Gatch, Indianapolis, Ind.

All those interested in attending the New York meeting should make their hotel reservations at once, and get in touch with Dr. James T. Gwathmey, Secretary, 40 East 41st St., New York City, for further particulars.

The Interstate Association of Anesthetists will meet in Toledo, Ohio, October 9, 10 and 11, in conjunction with the Mississippi Valley Medical Association. Details of this meeting will be published later.

THE LOUISVILLE SOCIETY OF ANESTHETISTS; THE KENTUCKY STATE BOARD OF HEALTH AND THE NURSE-ANESTHETIST.

The Supplement has already published some of the details regarding the preliminary circumstances and events that led up to the filing of the test-suit of *Dr. Louis Frank and Margaret Hatfield vs. the Kentucky State Board of Health* in the Chancery Division of the Jefferson County Circuit Court to determine the legal status of the nurse-anesthetist.

The final agreement in this test-case was for each side to file a single affidavit, each to include affidavits from any source from within or outside the state. The affidavit signed by Dr. A. T. McCormack, Sec'y. of the Board, showed the usual practice with reference to the administration of anesthesia by quoting from affidavits supplied by fourteen of the most prominent surgeons in Louisville. Further it included the results of a canvass of the American College of Surgeons, and affidavits from authoritative

(*Continued on page* 61.)

American Journal of Surgery

QUARTERLY SUPPLEMENT *of* ANESTHESIA *and* ANALGESIA

Surgery Publishing Co.

J. MacDONALD, Jr., M.D., President and Treasurer

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F. HOEFFER McMECHAN, A.M., M.D., *Editor*

Avon Lake, Ohio, U.S.A.

THE SCIENCE OF ANESTHESIA AND ANALGESIA.

In the present issue of the Supplement Dr. A. H. Miller makes a plea for accurate anesthetic records on the part of all anesthetists and the compilation of all available statistics by a Committee of the Organized Anesthetists. Such records and such statistics would serve a very definite purpose and would also result in disposing of many unwarranted clinical and empirical opinions, now held to the detriment of progress.

While standardized records would facilitate the compilation of statistics, at the same time such records would not always lend themselves to a solution of certain problems. Consequently, it is very desirable that even in keeping such routine records that the anesthetist should set himself to a more painstaking study of some definite and restricted and mooted question in the science or practice of anesthesia and analgesia.

In this way the individual anesthetist will accumulate protocols and records that will add something of definite value to the sum total of the science of anesthesia. The whole field of anesthesia and analgesia is wide open for review. Controversy, even thought it may become more or less polemic, makes a good clearing house for ideas. More attention to the physio-pathological phases of anesthesia and analgesia will redound to the benefit of all concerned. Johns Hopkins, Rush and the University of Indiana have shown conclusively that internes, under supervising anesthetists, can be successfully set to the investigation of certain scientific aspects of anesthesia, and the custom so pertinently set, should be followed more universally. Those who investigate such problems acquire the scientific spirit and habit, and become all the more valuable members of the profession, irrespective of what specialty they finally elect to pursue.—McM.

THE NURSE ANESTHETIST—CONTINUED.

In Ohio the efforts of the exponents of the nurse anesthetist to initiate a legislative amendment to legalize the nurse anesthetist have, so far, come to naught. The effort aroused the resentment of both the medical and dental professions. This agitation on the part of the nurse-anesthetist proponents also resulted in the chiropractors appearing before the Ohio State Medical Board and making a demand to be allowed to administer anesthesia for minor surgery and obstetrics.

In Pennsylvania the Bureau of Medical Education and Licensure, after considerable prodding, finally secured an opinion from the Deputy Attorney General, which held that nurses properly trained in anesthesia could legally administer anesthesia under the supervision of a licensed physician. Dr. J. M. Baldy has also announced, unofficially, that any medical man can take up residence in Pennsylvania and adminster anesthesia, under the supervision of a licensed physician, without complying with the usual requirements for reciprocity. This indicates the length to which the Pennsylvania Bureau is ready to go to support its subsidizing of the nurse anesthetist. The Bureau has been requested to give an official statement on the standards it has set for determining what constitutes proper training in anesthesia for nurses, but so far has not answered.

In Kentucky the Louisville surgeon who refused to abide by the opinion of the Attorney General and the decision of the Kentucky State Medical Association that the nurse anesthetist was illegal and unethical, has been accommodated with a test-case by the State Board of Health of Kentucky, which was decided in favor of the defendants by an opinion handed down by Judge Kirby of the Jefferson Circuit Court, March 10, upholding every point made by the State Board against the nurse-anesthetist. If necessary the case will now be taken to the Court of Appeals by the Attorney General for ratification.

It is interesting to note that both the Journals of the Indiana and Tennessee State Medical Associations have printed editorials discountenancing the nurse anesthetist. Further developments will be awaited with interest.—McM.

Newer Apparatus

Editor's Note: From time to time this Department will publish Illustrations and Technical Details of the Newer Apparatus for Anesthesia and Analgesia.

A NEW NITROUS OXID-OXYGEN-ETHER APPARATUS.*

KARL CONNELL, M. D.
Assistant Surgeon to Roosevelt Hospital; Associate in Surgery, College of Physicians and Surgeons.
NEW YORK CITY.

It is an honor for a surgeon to be invited to demonstrate his apparatus before the New York Society of Anesthetists.

Being afflicted by an inventive turn of mind, the field of mechanical aid to the administration of anesthetics has been a tempting one to dabble in, and since gases and vapors have not received the same mechanical measurement of dosage as have strychnin and morphin, I have invaded the field of ether administration in exact vapor tension dosage by devising the anesthetometer.

For five years past I have endeavored to build a gas oxygen apparatus that would enable one to obtain continuous control and knowledge over the flow of these gases.

The attachment to the anesthetometer which I demonstrated four years ago before this society, while yielding much valuable data on quantity and percentage of gas flow, has proved impracticable, as the ratio of the gases could not be rapidly changed, so experimentation along this line was abandoned. The method of sight feed through water, devised by Boothby, was experimented with when it first appeared, but attempt at measurement by the bubbling of gases from the open end of submerged tubes or through submerged slits and graduated holes submerged in water, glycerin, oil, and other media proved personally unsatisfactory, and because of the disadvantages of the bulk and splash of the liquid I ceased experimenting with this method after a few months.

At the Roosevelt Hospital for three years our anesthetists have used a miniature gas-oxygen apparatus weighing about one pound, which I devised. It depended for measurement of gases on a loose piston riding upward by gas pressure in a cylinder, and uncovering a calibrated slit for gas escapement, indicating by its upward excursion the gas flow with fair accuracy. The disadvantage of this piston apparatus was that the piston would at times stick in the cylinder, and several times we have presumed that the patient was getting oxygen when in fact the tank had run empty.

I searched the various loose piston devices in the United States patent office and concluded that none of these were sufficiently accurate and sensitive and at the same time reliable. I, therefore, turned for use in oxyacetylene blow

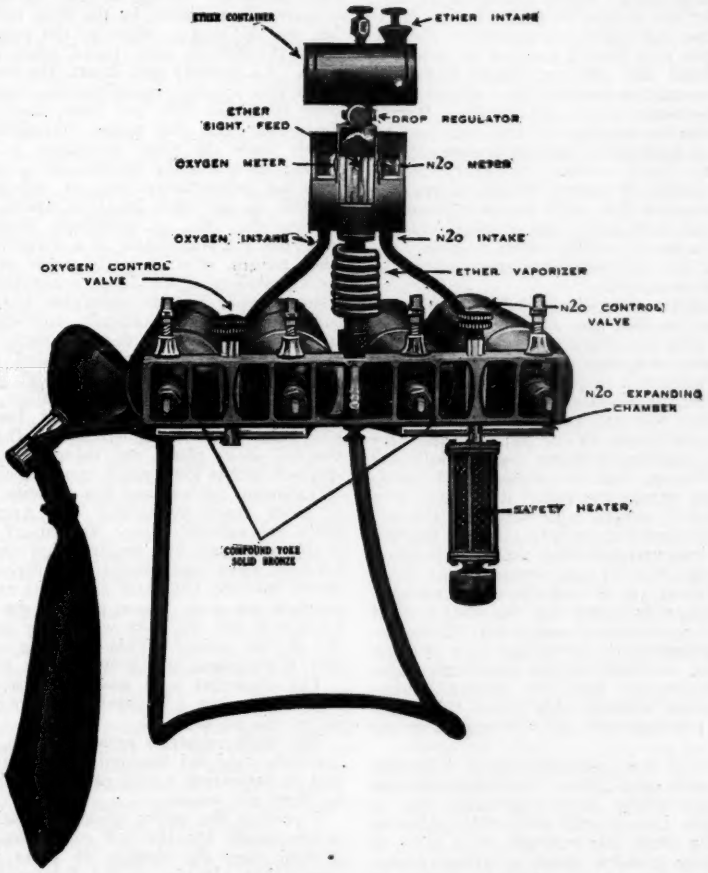

Fig. 1. Connell nitrous oxid-oxygen-ether apparatus assembled for use. Close-up of apparatus, showing the various details of construction and control.

* Presented at the meeting of the New York Society of Anesthetists, February 28, 1917.

torch, and elsewhere, to the idea of an unbalanced vane, substantially a disk, on the periphery of which gas impinges, actuating the disk against the force of gravity. By riding the disk on fine gold pinions in ruby jewel bearings, minute variations to the fraction of a teaspoonful of gas per minute were thus obtainable. On the periphery of this disk, a scale was mounted visible through a glass window in the housing and indicating the rate of gas flow much the same as the speedometer in your car indicates by figures on a disk how fast the car is going. The oxygen disk in the apparatus I exhibit, is calibrated to a flow in steps of one-tenth liter per minute from two-tenths to two and five-tenths liters per minute. The nitrous oxide disk is calibrated in steps of one liter per minute from two up to twelve liters per minute.

The reason for this range of calibration is as follows: It is found in practice that the average human subject became asphyxiated on an oxygen flow of less than five-tenths of a liter per minute, or, as you are more accustomed to express it, on a flow rated at seven and one-half gallons per hour. Indeed, even during induction it is found in practice that a continuous flow of eight-tenths of a liter per minute must be maintained. When symptoms of oxygen deprivation appear, the oxygen is gradually increased until the wants of that individual are satisfied. Usually this requires within the first fifteen minutes of administration, a flow of about one and two-tenths liters per minute. Later the flow may be increased to one and four-tenths or one and six-tenths liters per minute. Rarely does the flow have to be as rapid as two liters per minute, and then only in pathological states such as anemia, sepsis, and obstruction. Any larger volume, however, can be thrown through the meter, it merely ceases to register when the two and five-tenths liter mark has been passed.

Each meter, as you see, is about the size of a large watch. They could be made as fine as a lady's watch; in fact the first models were, but this necessitated a scale so fine in graduation as to be impracticable.

The nitrous oxid meter is also calibrated through a range of useful measurement for that gas, namely: It is found in practice when less than five liters per minute is administered, that the retention of carbon dioxid and other waste products becomes noxious, that the patient breathes excessively in depth and rate, the blood pressure rises and poor anesthesia is maintained, whereas, on a flow of from six to ten liters a minute, or ninety to one hundred and fifty gallons per hour, the breathing remains regular, full, and normal. The latter dosage has no physiological disadvantage, yet when one passes the eight liter mark, it is found that the quantity of oxygen must be proportionately raised, else the oxygen content in the tidal gases of respiration will become so impoverished that asphyxia becomes evident. Since the larger flow of gases possesses no physiological advantage except as it excludes inert nitrogen from a leaky face mask, it is found that the ideal flow of gas, considered from a combined standpoint of physiological and financial advantage, is a total gas flow of about seven liters per minute, of which oxygen constitutes eight-tenths to one and four-tenths liters per minute. When mixed by continuous flow with the tidal gases this yields the desired eight to fourteen per cent. of oxygen in the tidal intake.

The other features of the apparatus which I present have been gone into with equal care. The yoke presented tonight for the portable model, binds four tanks, two of gas and two of oxygen, into a solid unit. The tanks lie flat on their side on a chair, for example, or a table, in preference to the flimsy portable stand so often carried. The yoke is made of a solid phospho bronze casting. It weighs four pounds and is designed to stand a strain, at each yoke head, of six tons, which is more than it is possible to obtain by the screw device fastening the steel gas tanks in place. The control valves are seated in the bronze yoke and are adjustable with a thumb and forefinger, and the meters note the response to the various manipulations. Reducing valves of the spring type have been eliminated from this apparatus. The above plain pin valves have been adopted as more advantageous.

The pin valves for smooth continuous flow of the nitrous oxid must be kept slightly warm, otherwise this gas freezes and sputters. No extra heat at the oxygen valve is needed. Heat is added to the bronze expanded chamber tooled in the yoke and to the pin valve by an alcohol lamp, adopted as safer, more convenient, more reliable, and more efficient as a source of heat, than electric heaters. Most of the recorded accidents of inflaming ether vapor by mishaps to anesthetic devices have been caused by the spark of electric connections, and you well know the troubles of burned out connections. This lamp is a modified miniature miner's safety lamp. It is not possible to ignite from this lamp such combustible substances as ether, benzine, or illuminating gas. This I illustrate by pouring a teaspoonful of ether over the ignited and heated lamp. The ether does not ignite and the flame within the lamp merely flickers. I pour several ounces of ether over the lamp and instead of conflagration, the flame is extinguished because the combustion mixture within the lamp has become too rich and the flame cannot project itself outside the wire gauge. Thus we have a safe and reliable heating device. The novel feature of this lamp is the copper conductor of heat whereby the minimum of flame yields direct to the yoke the maximum of heat.

As a final feature of the apparatus, I desire to demonstrate the ether mixing device. Ether is fed into the gas mixture when desired by the drop method. I have found that the addition of ether by the method of shunting the gases through or over liquid ether has many disadvantages. As Boothby has shown, the amount of ether taken up by this process can neither be approximated nor regulated to within fifty per cent., and may vary widely at times, nor can the same concentration be maintained evenly since the ether is subject to certain rigid vapor tension laws, and the tension differs so widely with variations of temperature due to refrigeration. The drop method, on the other hand, is visible, and while censured because the drop as ordinarily done from a crude oil cup, does not stay evenly at a given rate, yet this method, when refined, offers a fairly stable and accurate basis for the calculation of the vapor concentration delivered. The ether dropper in this apparatus has been made smooth and regular by mechanically correcting the control pin, from the type accustomed to handle oil and by the employment of a horizontally placed tank, thus avoiding marked diminution in the vertical head of liquid as it becomes consumed. The liquid ether drops from a standardized ball tip through a sight feed into a spiral coil when the ether is vaporized into the gases. While this fine coil looks like a toy, being only an inch in diameter and two inches long, yet it contains eighteen square inches of radiating surface and has capacity to vaporize 180 mm. of ether vapor by tension into a gas flow of eighteen liters per minute. Since 80 mm. of ether vapor tension is the maximum one required for induction of ether relaxation when superimposed on nitrous oxid-oxygen anesthesia, this coil possesses an excess capacity of about four hundred per cent. From the coil the gases and vaporized ether flow into the yoke when they are warmed for delivery to the patient. This warming is for sentiment, not that it possesses any demonstrable advantage.

The apparatus as a whole weighs about seven pounds, and as you note, I brought the three parts to this meeting in my hip pockets.

The mask possesses novel features only in being a very carefully modeled anatomical fit, with suction type joint, and in possessing a spill calibrated for continuous outflow in liters per minute.

I present the entire apparatus with a certain pride of achievement, for the aid of accurate measurement and control over the dosage of these intangible anesthetic agents has already, at the Roosevelt Hospital with the first rough model, enabled us to direct the novice anesthetist as to limits of dosage, and has enabled him to obtain, on the first trial, a perfection of anesthetic control hitherto achieved only by highly trained expert.

ROOSEVELT HOSPITAL.

THE AMERICAN ASSOCIATION OF ANESTHETISTS WILL MEET IN NEW YORK CITY, JUNE 2, 1917 AT THE HOTEL McALPIN FOR ITS SCIENTIFIC SESSION AND ANNUAL BANQUET. CLINICS WILL BE HELD JUNE 4-5, IN CONJUNCTION WITH THE AMERICAN MEDICAL ASSOCIATION.

Society Proceedings

(*Continued from page 57.*)

sources throughout the United States. Arguments were included to show why a knowledge of medicine was necessary in order to properly and safely administer anesthetics.

While this first affidavit seemed sufficient to meet and refute the original affidavit of the plaintiffs, a few days before the hearing, the plaintiffs filed a supplementary affidavit, alleging that "In Kentucky, a number of public and private hospitals, anesthetics are administered by trained nurses; that this is true of the Norton Memorial Infirmary and the Children's Free Hospital, Louisville; Spner's Hospital, Dayton; Illinois Central Hospital, Paducah; St. Joseph's and Good Samaritan Hospitals, Lexington, and it is not a fact that the practice has been abandoned in such hospitals. In addition anesthetics have been administered by a trained nurse in the Jewish Hospital, St. Joseph's. and at St. Mary's and St. Elizabeth's Hospitals, Louisville. In the Louisville City Hospital, which is very much the largest in the state, anesthetics, in practically all surgical cases are administered by internes, who are not licensed physicians, and it is the usual and daily practice for medical students who are not licensed, to administer anesthetics."

Other statements were included in the plaintiff's supplementary petition to discredit that of the State Board, and the paper, read by Dr. J. M. Baldy of Philadelphia at the meeting of the American Gynecological Society, was incorporated in full. The plaintiffs also alleged that no deaths had ever been heard of as occuring while a nurse was the anesthetist, while many deaths were on record while physicians were the anesthetists.

In answer to this supplementary affidavit, the State Board secured affidavits from two resident physicians of St. Joheph's Hospital, the present and former one, stating that in only two instances during the past two years had anesthetics been administered by anyone except physicians and that in the two instances mentioned, the plaintiffs themselves were the operator and anesthetist. An affidavit was also obtained from Dr. Fowler, Supt. of the Louisville City Hospital, who stated that: "Anesthetics are always administered either by one of the resident physicians, all of whom are graduates of 'Class A' medical colleges, or by one of the members of the visiting staff of anesthetists. . . Members of the Junior and Senior classes are permitted to witness operations and make observations, but never has a medical student or a nurse administered a general anesthetic, at the Louisville City Hospital."

Dr. Horine also submitted an affidavit stating that he was familiar with the work done at the Louisville, Jewish, St. Anthony's, Deaconess', St. Mary's and St. Elizabeth's, Children's and Norton Hospitals, and that physicians were always the anesthetists, except in the cases operated on by the plaintiffs. A statement was also secured from a young physician, who had just returned from three years' service at the Mayo Clinic, where he had witnessed two deaths, while nurses were administering anesthesia, and other fatalities of a similar character were submitted.

The supplementary affidavit submitted by the State Board thus refuted that of the plaintiffs in every detail.

The hearing of the test-case was before Judge Samuel B. Kirby, in the Chancery Division of the Jefferson Circuit Court, Louisville, February 17, and lasted a little over two hours. The two affidavits were filed with the briefs and then the Judge requested an oral statement from both sides. The attorney representing the plaintiffs attempted to inject the Organized Anesthetists into the matter by saying that they desired to create a monopoly, but Judge Kirby very courteously advised him that this attack was irrelevant and without bearing in the case at bar. The attorney for the State Board based his argument mainly on the legal questions involved. The Judge then took the case under advisement and delivered his opinion on March 10, holding that: "The trained nurse, (in administering anesthesia) is performing the function of a physician and in the language of the statute is discharging the duties usually performed by a physician." The Judge admitted that the nurse in this instance was trained, but "as contended by the defendant, if she be allowed to perform this service without having complied with the law, any other trained nurse, who has not her skill or ability or training or other qualifications and however ignorant, may perform similar service. . . It is the example, which the State Board of Health is objecting to."

There is every prospect that the test-case will now be taken to the Supreme Court, (Court of Appeals), for final adjudicating. In this event, the Attorney General of Kentucky, the Hon. M. M. Logan, is prepared to present a far stronger argument for the State Board, than was presented at the first hearing, with the additional backing of some of the highest medical and educational authority.

It is to be hoped that this favorable decision in Kentucky will enable State Boards in other states to put a stop to the activities of certain surgeons and hospitals in attempting the disintegration of the practice of medicine to the detriment of public health and safety.

APOTHESINE, A NEW LOCAL ANESTHETIC.

Carroll W. Allen, M. D.

New Orleans, La.

It it with much pleasure that we note an effort on the part of our chemical manufacturers to provide us with drugs, the original products of their own laboratories and make us independent of foreign sources, which have so long been our only source of supply. Apothesine, recently presented in experimental quantities by Parke Davis & Co., is a synthetic product of their laboratory. Chemically, it is the cinnamic ester of grammadiethylamino propyl alcohol hydrochlorid. Apothesine occurs in the form of small snow-white crystals, having a melting point of approximately 137° C. It is easily soluble in alcohol; very soluble in water; and slightly soluble in acetone and ether.

During a clinical experience of several months, I have been very favorably impressed with the anesthetic properties of this product and have performed hernia, hemorrhoid, fistula, varicocele, circumcision and plastic operations. I have uniformly used a ½ per cent. solution in 4 per cent. sodium chlorid with five drops of adrenalin chlorid solution, 1 to 1000 to each ounce. The anesthesia has been complete in all cases and has invariably lasted in excess of an hour. No immediate or late irritating effect was noted and the wounds healed as well as after the use of any other anesthetic solution. No toxic or other unpleasant immediate or after effects were noted, although large quantities were purposely used to determine this point; as much as four ounces in one case, which represented nearly ten grains of the drug. This limited experience, with the absence of other data, is not sufficient to give it its proper place among local anesthetic agents. Data regarding its toxicity are not obtainable, but from my experience it must be very low. In this connection, the manufacturers state that its toxicity is not greater than that of novocain and considerably less than that of other anesthetics commonly employed.

I have not been able to obtain any more exact information than the above and the time at my disposal does not permit of further observation.

The following are the results of injections made upon myself in studying its action. The preparation experimented with was in liquid form put up with a saturated solution of chloretone for purposes of preservation, which I believe is a mistake, as noted later. The solution was first boiled for ten minutes. Intradermal wheals were made in my skin at various points, using for each injection one c.c. of solution.

1. One per cent of apothesine, injection 9:55 p. m., slight negligible burning if injected too rapidly, which immediately subsided; no sensation if slowly injected. Immediate anesthesia. Within about ten minutes slightly pale area had developed around wheal and center became slightly pink. After fifteen to twenty minutes slight itching sensation. 10:30 p. m. pink center has become more marked. 11:10 anesthesia still complete. 11.25, returning sensation. 11:55, nearly normal sensation. 12:30, not yet

quite normal. Complete anesthesia for one hour and fifteen minutes. Pink center to wheal still persists, with a very slight suggestion of infiltration in surrounding tissues.

2. One per cent apothesine, five drops of adrenalin, 1 to 1000 to the ounce, injection 10:02, results similar to last injection, except that pale area surrounding wheal was slightly more marked, with pink center more pronounced. 12:15, anesthesia still complete. 12:30, returning sensation. Complete anesthesia one hour and fifteen minutes. Subsequent appearance of area same as with 1 per cent without the adrenalin.

3. One-half per cent apothesine, sodium chlorid 4 per cent; injection 10:07, no sensation during injection. Immediate anesthesia. Pink center and pale surrounding area as with other injections. 10:35, complete anesthesia. 10:40, returning sensation. 10:45, near normal. 11 o'clock, normal. Thirty minutes' complete anesthesia.

4. One-half per cent of apothesine, adrenalin solution 1 to 1000, five drops to the ounce. Injection 11 o'clock. Immediate anesthesia. Appearance same as above. Forty-five minutes' complete anesthesia.

5. One-half per cent apothesine, sodium chlorid 4 per cent, adrenalin solution 1 to 1000, five drops to the ounce. Injection 10:13. 11:30, complete anesthesia. 11:37, returning sensation. 11:50, nearly normal. Complete anesthesia one hour and fifteen minutes. Appearance of injected area same as above.

6. One-fourth per cent apothesine. Injection 10:33. No sensation. Immediate anesthesia. 10:55, complete anesthesia. 11:05, returning sensation. 11:15, nearly normal. 11:28, normal. Pink center and pale surrounding area less marked than with the stronger solutions.

7. One-fourth per cent apothesine, 4 per cent sodium chlorid. Injection 10:38. No sensation. 11:15, complete anesthesia. 11:19, returning sensation. 11:35, nearly normal. 11:50, normal. Thirty-seven minutes' complete anesthesia. After appearance same as above.

8. One-fourth per cent apothesine, adrenalin solution 1 to 1000, five drops to ounce. Injection 11 o'clock. 11:40, complete anesthesia. 11:45, returning sensation. Forty minutes' complete anesthesia. After appearance same as above.

9. One-fourth per cent apothesine, 4 per cent sodium chlorid, adrenalin solution 1 to 1000, five drops to ounce. Injection 11:13. 11:50, complete anesthesia. 12 o'clock, returning sensation. 12:35, nearly normal. Thirty-seven minutes' complete anesthesia.

The next day, a slightly reddish punctate spot marks the site of the wheals. This is more marked in those in which the stronger solutions have been used and slightly more so where adrenalin had been added. Slight soreness and itching in all spots when manipulated, but not noticeable without manipulation. In the two 1 per cent injections a very slight infiltration of the tissues noticeable. The frequent examination of the areas in which they were stuck by a needle every few minutes during the period of the examination should be considered in drawing conclusions from their appearance. The anesthesia in all cases was immediate and complete and disappeared very slowly. It was often longer than one-half hour from the time returning sensation was first noted until it appeared normal. The pink appearance in the center of the wheal in the case of the stronger solutions persisted for forty-eight hours, but no similar reaction had been noticed in the clinical use of the drug, although the wound had been closely watched. To determine the cause of this reaction two injections were made into my skin with solutions, prepared by tablets of the same drug which did not contain chloretone. In each case there was no sensation during the injection and the central pink area and surrounding pale areola were less marked and disappeared after a few hours. Otherwise, the resulting anesthesia and its duration were the same. The site of injection, if disturbed by manipulation, gave a slight itching sensation, otherwise it was not noticeable.

To test the effect of chloretone when injected alone, I injected my forearm with ten minims of a saturated solution of chloretone, 8 per cent, the same strength with which the apothesine is put up; slight burning occurred if injected too rapidly. When tested with a point of a needle for anesthesia, it was found to be completely anesthetic only in the center of the wheal and wherever the needle was entered, a red punctate spot appeared immediately and remained permanently. Otherwise, the wheal presented no change in appearance from the surrounding tissue. Anesthesia lasted about forty minutes' only in the center; the periphery quickly returning to normal.

I am consequently forced to the conclusion that much of the after appearance of the injected areas in these experiments must be due to the presence of chloretone in the solution and accordingly suggest that this method of preparing the solution be changed, as it probably will, and a drug marketed in powder form, as is the case with other local anesthetics.

In conclusion, I wish to state that I believe this preparation has a decidedly useful future and should be given a fair and impartial trial. In my own observation it compares, at least in its anesthetic producing properties and its low degree of toxicity, very favorably with novocain.

ANESTHESIA: SUGGESTION BEARING UPON ITS STANDARDIZATION FOR TEACHING PURPOSES.*

PALUEL J. FLAGG, M.D.

Any man who administers anesthetics is at some time or other called upon to impart his knowledge to others who are being initiated into this special branch of the practice of medicine. In order to get close to the student, we must explain the subject in simple words before we allow ourselves to become technical. It is perfectly easy to lose our audience by employing unfamiliar terms, or terms which mean little to the average man. For example, take the term, "vapor tension." What does that mean to the man whose last intimate acquaintance with physics was five or more years ago? The incidental employment of such a term may pass, but if we proceed to make use of it to illustrate or compare, our audience is left behind. According to the general meaning of the term, teaching is not an effort to ventilate our opinions, but to impart them to others, and this can be done only by being sure that the terms which we employ are perfectly familiar to all.

Men who are specially interested in a particular subject are prone to ignore or slur over the wearisome and simple fundamentals which brought them to their present point of view. As a consequence, the student who has missed the fundamentals cannot grasp or appreciate that which is being placed before him. In teaching the art of anesthesia, we are prone to depend upon the information which the student is supposed to have gained in his materia medica and therapeutics. In describing the signs of anesthesia, we speak of them as occurring during certain stages. We speak of the excitement of the first stage, of the dilated pupil of the third stage. The term, preanesthetic stage, is often heard. It is said that such and such an operation can be done in the preanesthetic stage, during which the patient *is* yet *is not* anesthetized. We have been brought up upon the term, surgical anesthesia. This is thought to be a very definite stage, in spite of the fact that anesthesia sufficient for the opening of a boil is a very different thing from that which is needed for a laparotomy. If we can establish a classification of the stages of anesthesia which can be used as a basis for all anesthetic agents administered by all methods, we may speak to the uninitiated and to one another in a manner to be understood.

The classification of the stages of anesthesia which follows might be called a clinical classification, or a classification combining the findings of the physiological laboratory and the operating room. This classification is not only easily grasped, but is equally easy to retain and demonstrate.

GENERAL, LOCAL, AND SPINAL OR MIXED ANESTHESIA.

These form the three primary divisions, and are based upon the extent to which the nervous system is anes-

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thetized. In general anesthesia both the central and the peripheral nervous system is anesthetized; in local anesthesia only the peripheral nervous system is involved. In mixed or spinal anesthesia, part of the central nervous system (the spinal cord) and the peripheral nervous system are under the influence of the anesthetic.

We recognize two degrees of general anesthesia, complete and incomplete. These degrees are characterized by the presence or the absence of complete muscular relaxation. In a complete general anesthesia, the relaxation is universal; in an incomplete general anesthesia, relaxation is partial or absent.

A complete general anesthesia consists of three stages: 1. Induction; 2, maintenance; 3, recovery. These three stages may readily be represented by a curve (Fig. 1). They are present and demonstrable with all anesthetic agents and with all methods which yield complete muscular relaxation.

The stage of induction begins with a loss of consciousness and ends with the onset of complete relaxation.

The stage of maintenance begins with the completion of relaxation and continues until the administration of the anesthetic is permanently reduced with a view of bringing the patient back to consciousness.

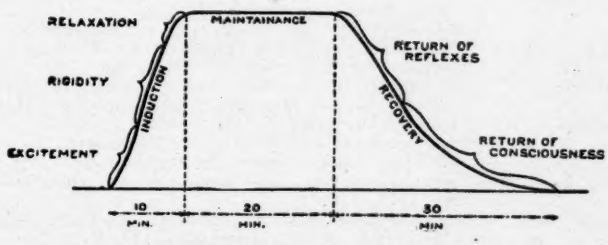

Fig. 1.—Curve of complete anesthesia; *a*, excitement; *b*, rigidity; *c*, relaxation; *d*, return of reflexes; *e*, return of consciousness; *f*, induction; *g*, recovery. (Courtesy J. B. Lippincott Co., *Art of Anesthesia*.)

The stage of recovery begins when the administration of the anesthetic is permanently reduced with the view of bringing the patient back to consciousness, and ends with the return of consciousness. Induction anesthetizes the patient; maintenance keeps him anesthetized; recovery returns him to consciousness.

The stage of induction consists of three periods: 1. Excitement; 2, rigidity; 3, relaxation.

The stage of maintenance is not subdivided, but two varieties may be observed: *a*, Constant maintenance; *b*, variable maintenance.

Constant maintenance is obtainable only where a special device is employed which will deliver to the patient a large volume of ether vapor of a definite percentage under pressure; such an apparatus as the Connell anesthetometer, for example.

All other methods of anesthetization yield a *variable maintenance.*

The stage of recovery is naturally divided into two periods: 1. The return of the reflexes; 2, the return of consciousness.

The end of the first period comes about in the operating room, while the patient is under the immediate supervision of the anesthetist. The end of the second period usually takes place after the patient has reached his bed, and has passed into the nurse's care.

Two types of recovery are easily recognized: 1. Recovery by crisis; 2, recovery by lysis.

The first type is best characterized by the recovery which we see in a gas oxygen anesthesia; the second is the usual recovery seen in a prolonged ether anesthesia.

This very simple classification of the stages of anesthesia may be used as a basis for the discussion of any anesthetic agent administered by any method.

Where a detailed study is desirable, we may attack the subject by considering the evidences, the causes, and the control of the periods of excitement, rigidity, and relaxation; for example, the causes of the period of excitement:

Physiological effects of ether.
Lack of preparation or faulty preparation.
Alcoholism.
Temperament.
Overconcentration of the anesthetic.
Excessive smoking.
Sexual.
Previous unsatisfactory anesthesia.
Nasal obstruction, almost the rule in children.
Failure to understand what anesthesia means.
Excessive fear.

Remarks which the patient hears before losing consciousness.

The curve which represents complete general anesthesia forms a basis upon which we may become familiar with the idea of vapor tension or anesthesia by the percentage method.

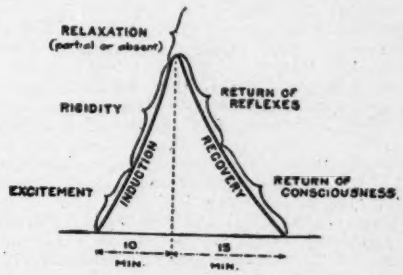

Fig. 2.—Curve of incomplete anesthesia; *a*, excitement; *b*, rigidity; *c*, relaxation; *d*, return of reflexes; *e*, return of consciousness; *f*, induction; *g*, recovery. (Courtesy of J. B. Lippincott Co., *Art of Anesthesia*.)

Where such a classification is employed, the signs of anesthesia may be definitely charted. We may speak of the behavior of the pupil under ether during the stage of induction, maintenance, and recovery with the assurance that the time specified will not be misunderstood. We may speak of the complicating effects of morphine upon the same basis. The detailed effects produced by rebreathing with various anesthetics are thus quite easily identified.

It will be seen from the foregoing that muscular relaxation is a test of the worth and efficiency of the general anesthetic. An anesthetic which will not bring about good relaxation, even though it is safe, can never be other than a method of choice in selected cases. We must not forget that the anesthetic is but a means to an end, not an end in itself; that the inexperienced surgeon, the man who does not object to rigidity, will not learn to do good work, that the experienced surgeon cannot do his best work in the face of muscular rigidity.

In upholding the cause of relaxation, we do not intend to overshadow *safety*, immediate or remote. We speak of this quality as *an* essential, not as *the* essential. We do not intend to discountenance gas oxygen or to exalt chloroform. What we wish to do is to emphasize the necessity of relaxation where indicated, and to point out the fact that the routine anesthetic must be capable of bringing about this effect.

In conclusion, then, we offer this clinical classification of the stages of anesthesia in the hope of simplifying the technical subjects which we present for the consideration of one another.

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